A Dissertation on

AN OBSERVATIONAL STUDY ON MUCOCUTANEOUS MANIFESTATIONS OF TYPE 1 AND TYPE 2 DIABETES MELLITUS

Submitted to

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CERTIFICATE BY THE INSTITUTION

This is to certify that **Dr. T.JAYA PACKIAM,** Post - Graduate Student (May 2012 TO April 2015) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**AN OBSERVATIONAL STUDY ON MUCOCUTANEOUS MANIFESTATIONS OF TYPE 1 AND TYPE 2 DIABETES MELLITUS"** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr. M. G. R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2015.

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DECLARATION

I, **Dr. T.JAYA PACKIAM,** declare that I carried out this work on "**AN OBSERVATIONAL STUDY ON MUCOCUTANEOUS MANIFESTATIONS OF TYPE 1 AND TYPE 2 DIABETES MELLITUS**" at the Medical wards, Diabetology OP & Dermatology OP of Government Stanley Hospital during the period February 2014 to September 2014. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu DR. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M. D. Degree examination in General Medicine.

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CONTENTS

ABBREVIATIONS

CDC	-	Centers of disease control
DM	-	Diabetes Mellitus
NEG	-	Nonenzymatic glycosylation
TcPo2	-	Transcutaneous oxygen pressure
AN	-	Acanthosis nigricans
IBW	-	Ideal body weight
SLS	-	Scleroderma-like syndrome
LJM	-	Limited Joint syndrome
AEGs	-	Advanced glycation end-product
MEO	-	Maligant External otitis
CSII	-	Continuous subcutaneous Insulin infusion
RCM	-	Rhino cerebral mucormycosis
NL	-	Necrobiosis lipoidica
NLD	-	Necrobiosis lipoidica diabeticorum
GA	-	Granuloma Annulare
PAS	-	Periodic acid schiff
PCT	-	Porphyria cutanea tarda
APD	-	Acquired perforating dermatosis
RPC	-	Reactive perforating collagenosis
NME	-	Necrolytic migratory erythema.

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INTRODUCTION

Diabetes Mellitus is a group of metabolic disorders with different subtypes of diabetes with hyperglycemia Regardless of its etiopathology, diabetes progresses through several clinical stages. It may present with characteristic symptoms or individuals may remain asymptomatic for a very long time.Consequently, diabetes is often diagnosed by an abnormal routine blood test by then some complication has already set in.

Diabetes mellitus is a group of metabolic disease. It is due to insulin secretary defect and insulin action or both. Dysfunction or organ failure occurs with hyperglycemia. Organs affected are kidney, nerves, eyes, heart and blood vessels, skin.

CLASSIFICATION

Two types:

Type 1 Diabetes – lack of insulin secretion.

In Honeymoon remission phase patient will return to normoglycemia even after ketoacidosis. It may require insulin. Eg: type 1 diabetes mellitus occur in pregnancy period.

Type 2 Diabetes – due to resistant to insulin and insulin secretory defect.

Diabetes develops with congenital defect in beta cell function, congenital defect in insulin action, exocrine pancreas defect. Depends upon the disease process, the degree of hyperglycemia varies.

ETIOLOGICAL CLASSIFICATION OF DIABETES

1. Diabetes mellitus Type 1
    a. Immunologically related
    b. Unknown cause
2. Type 2 diabetes mellitus
3. Other specific types
    a. Genetically mediated defects in function of Beta cells-MODY type 1 to type 6 mitochondrial diabetes
    b. Genetically mediated defects in action of insulin – Insulin resistance type A
    c. Lipoatrophic diabetes
    d. Pancreatic disease
        (i) Fibro calcific pancreatitis
        (ii) Pancreatectomy
        (iii) Cystic fibrosis
    e. Endocrinopathis
        (i) Acromegaly
        (ii) Cushing's syndrome

(iii) Phaeochromocytoma

(iv) Hyperthyroidism

f. Drug induced

Glucocorticoids

Thyroid hormone

Diazoxide

Thizides

Dilantine

Vacor,pentamidine, olanzepine,rifampicin

g. Infections

Congenital Rubella

Cytomegalovirus

Mumps

h. Uncommon forms of diabetes

"stiff-man" syndrome -Due to antibodies against insulin receptor

i. Genetic syndrome association

Down's syndrome

Turner's syndrome

Klienfelter's syndrome

Myotonic dystrophy

Praderwilli syndrome.

4. Gestational diabetes

PATHOGENESIS

TYPE 1 DIABETES MELLITUS

Type 1 diabetes is of autoimmune origin and is related to a genetic – environmental interaction of human leukocyte antigen genes with an unknown environmental trigger which could vary from viruses to cow's milk protein. Autoimmune islet cell destruction occurs which leads to release of antigens and the production of multiple antibodies as an epiphenomenon which are in fact detectable prior to the onset of diabetes in greater quantities and decline in titer after several years of diabetes have transpired.The genetic component of Type 1 diabetes is stronger than that of type 2 diabetes contrary to that of traditional thinking.

Genetic Factors

Type 1A diabetes is due to multiple gene polymorphisms. In genes, apart from major histocompatibility complex (MHC), larger effect is due to HLA alleles; followed by insulin gene polymorphism and lymphocytes specific tyrosine phosphate.HLA DR3 & DR4 is present in most of type 1 Diabetes mellitus. Other candidate genes include CTLA-4.IL2RA, SH2B3, etc.

Autoimmunity

Initiation and progression of autoimmune diabetes mellitus is due to auto antigen is present in pancreatic Beta cell. These auto antigens include

insulin, Glutamic-acid decarboxylase(GAD), insulinoma-associated protein 2 and the autoantigens is znT8, a zinc transporter of islet beta cells. In initiation of injury, exact initiating auto antigen is not known. The autoimmune response in type 1 diabetes affect other organs like thyroid, adrenal, tissue tranglutaminase (celiac disease).There is also an increased evidence of cell mediated immunity.

Environmental Factors

Virus infections affect Beta cells by initiating autoimmunity. The candidate viruses are Coxsackie and certain enteroviruses. Diet is also important factor for development of T1 diabetes mellitus. Those are albumin in cow's milk, most of infant formulas.

TYPE 2 DIABETES MELLITUS

Type 2 diabetes is classified as a "complex disease". It is a disorder of polygenetic in origin and is strongly influenced by environment factors (in contrast to thalassemia which is a "simple" disease that has a genetic origin not influenced too much by environmental factors).Type 2 diabetes was traditionally marked as a disease precipitated largely by insulin resistance due to weight gain and lack of physical activity.However most recent research indicates that it is of multifactorial origin.

Genetic Factors

New diabetes susceptibility loci are identified by Genomic analysis. Various genes involved in pancreatic development, beta cell function, insulin synthesis, insulin release and action and these are found to be associated with increased risk of type 2 diabetes.The one gene which seems to be important in several populations for an early secretory defect right across the board is Transcription factor 7 like (TCF7L2) which regulates beta cell survival and function. Transcription factors that contain high mobility domains it binds to Beta carotenin & activate receptors, this complex initiate expression of target genes.

KCNJ11: Sulfonylurea receptor is down streamed by islet ATP - sensitive gene potassium channel.Spans 1 kb and is located directly downstream of the gene encoding the Sulfonylurea receptor.Adipocyte differentiation is by transcription factor, PPAR gamma 2, the gene that encodes the peroxisome proliferator – activated receptor, itself a target for medications that lower insulin resistance: the P12A polymorphism in PPAR gamma had been conclusively associated with common type 2 diabetes.Other candidate genes – beta 3- adrenergic receptor, calpain 10, HNF1 beta and IGF2BP2 are the genes found to be associated with type 2 diabetes. Type 2 Diabetes mellitus is also associated with polymorphism candidate genes.

Environmental Factors

Lifestyle

The major precipitating factor is environmental which encompasses diet and sedentary lifestyle. Ironically , high calorie dense foodstuffs may actually cost less when compared to healthy food items like fruits and green vegetables.The lack of physical activity is due to improved forms of motorized transport,escalators and elevators; professions which entail little physical activity and encourage a sedentary such as in the software industry and other similar jobs. Communication facilities like mobile telephony; entertainment facilities like television and computer/video games are now available at a low cost. All in all, enormous lifestyle changes have occurred in the last three decades even in developing countries increasing the propensity of the problem.

Role of obesity and inflammation

Obesity is the cause for peripheral resistance to insulin. It decreases sensitivity of Beta cells to glucose. It decreases insulin mediated glucose uptake. Weight loss reduces blood sugar level.

Inflammation is mediator to obesity, Diabetes and atherosclerosis. Type 2 Diabetes Mellitus is also associated with increased levels of C reactive proteins, IL-6, PAN-1 & WBC. Adipokines are responsible for inflammatory activity which are released by adipokines .These adipokines

include leptin, adipocytes, tumor necrosis factor, resistin, obestatin, upcoupling protein -2 and retinol binding protein - 4

Role of intrauterine development

The relationship between birth weight and subsequent risk of type 2 diabetes is U shaped. Both low and high birth weight are associated with increased risk of type 2 diabetes in adulthood, though this is well proven with low birth weight. Increased risk of type 2 Diabetes mellitus associated with higher birth weight and premature babies. Low birth weight, by multiple mechanisms may lead to a greater propensity for diabetes mellitus later in life which include insulin resistance as well as insulin secretory defect.This could be due to increased peripheral insulin resistance, especially in muscles and reduced hepatic insulin sensitivity. Additionally defects in GLUT-4 and insulin secretion have been identified in those born low birth weight. A secretory defect in the beta cell may be present from a fairly early point of time in life.Increased risk of diabetes is associated with higher birth weight (>4.0 kg). Children born prematurely, irrespective of their birth weight, may be at increased risk for type 2 diabetes and other disease of adulthood associated with insulin resistance.

DIAGNOSIS OF DIABETES MELLITUS

Blood glucose is the important for diagnosis of diabetes mellitus. Three ways to diagnose diabetes are possible, and subsequent days random blood

sugar level is to be seen.75 gm OGTT is more sensitive and rarely performed. Because of its ease, patientsacceptability and lower cost, measurement of fasting plasma glucose is the preferred diagnostic test.

Impaired fasting glucose -Fasting glucose 100 – 125mg/dl

Impaired glucose tolerance - 2 hour post-load/postprandial glucose 140-199 mg/dl

CRITERIA FOR THE DIAGNOSIS OF DIABETES MELLITUS

Symptoms of hyperglycaemia and a causal plasma glucose > equal to 200 mg/dl. Casual is defined as any time of day without regard to time since last meal. The classic symptoms of hyperglycemia include polyuria, polydipsia and unexplained weight loss.

(Or)

Fasting >equal to 126 mg/dl. Fasting is defined as no caloric intake for at least 8 hours.

(Or)

2-h plasma glucose > equal to 200 mg/dl during an OGTT or postprandial. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

This is the basis of the definition of fasting plasma glucose levels being more than 126mg/dl as a defining point in diabetes.

EPIDEMIOLOGY OF DIABETES MELLITUS

TYPE 1 DIABETES MELLITUS

Type 1 diabetes previously called insulin-dependent or juvenile diabetes. It is caused by destruction of beta cells. It is mediated through immune mechanism, leads to lack of insulin secretion and insulin deficiency. Type 1a (autoimmune form) diabetes (T1aDM) is precede by a subclinical period of T-cell- mediated autoimmune destruction of beta cells. T- cell mediated beta cell destruction results in auto antibodies formation. In children it is usually rapid onset of symptoms and dependence on exogenous insulin for survival.

NATURAL HISTORY OF TYPE 1a DIABETES

The natural history of type 1a DM includes four distinct stages:

1. Autoimmunity to Beta cells with insulin secretory defect.
2. Appearance of clinical diabetes
3. Transient recovery
4. Once diabetes is established it leads to acute and chronic complications and death in premature.

Preclinical Beta Cell Autoimmunity

Type 1a DM is due to autoimmune destruction of Beta cells in genetically susceptible host to environment. Antibodies against specific beta cell antigens, such as glutamic acid decarboxylase (GAD) or ICA512.

Family History

The prevalence in first degree relatives of type 1 diabetic persons is increased 3-5 times, compared to that in school children with no family history of T1DM. The risk of T1DM among the relatives is 20 times higher than in the general population.

Candidate Genes

The HLA-DR2, protects type 1 Diabetes Mellitus. it is found in about 15% of GAD and young relatives of IAA positive patients. However, over 90% of those strongly and/or persistently ICA positive are HLA-DR3 or 4, similar to T1DM patients.

ENVIRONMENTAL FACTORS

Viruses

Viral infections instigate autoimmunity. Two infections with similar viruses are responsible for type 1 diabetes mellitus. Viruses are mumps, rubella, measles, chickenpox, coxsackie, ECHO4 and rotavirus infection. Newborns and infants are particularly likely to develop a persistent infection and among patients with congenital rubella syndrome, 70% have ICAs.

P2-C protein of coxsackie virus and the GAD protein may be responsible for autoimmunity.

Dietary Factors

The intestinal barrier is compromised when it is exposed to cow's milk bovine serum or beta casein. It is the important cause for autoimmunity.

The association between cow's milk and autoimmunity could be due to the effect of beta – casein immune stimulating hexapeptide. It is present in Bos Taurus cows.

CLINICAL ONSET OF T1DM

25-45% of Type 1 DM patients are younger than 20 years. Severe manifestations occur with DR4 allele, younger age, female gender, no family history, lower socio economic status. If 90% of islets are destructed, type 1 DM will present as severe manifestations. In the age of 7- 14 years, 60% of beta cells are destructed. If the age of patients is more than 14 years, the beta cell destruction is 40%.

Symptoms of diabetes mellitus polyuria, polydipsia, and weight loss 3-5 weeks prior to diagnosis.

Age

Due to alteration in infection pattern and increase in insulin resistance, type 1 diabetes mellitus peaks at 2yrs, 3-5yrs, 9 – 15 yrs.

Gender

T1DM occurs equally in males and females. Peak incidence in females is 1-2 years earlier than males.

Viruses

Herpesviruses, mumps, rubella, and retroviruses have been implicated. There is also increasing incidence of T1DM in patients with congenital rubella syndrome. In T1DM, congenital rubella syndrome is also responsible. In postnatal, rubella exposure to the MMR vaccine causes T1DM. The incubation period of T1DM in CRS patients is 6-21 years.No increase in risk of diabetes or pre diabetic autoimmunity with routine childhood immunization.

DIETARY FACTORS

There is also association between T1DM and nitrates and nitrites present in water.

REMISSION ("HONEYMOON PERIOD")

Those who are presenting in older age and less severe initial presentation, and low or absent ICA or IA are associated with deeper and longer remission. Younger children those who are having HLA DR3, DR4, phenotype leads to destruction of beta cell occur within 3 years of diagnosis. In older patients it is slower and partial. In 18% of patients, function of beta cell is preserved even 10yrs after diagnosis.

COMPLICATIONS

Acute complications of T1DM (diabetic ketoacidosis, low blood sugar, and infection.). For renal failure,vision loss, amputation, diabetes is the important cause.Other reasons are cardio vascular disease and premature death.

TYPE 2 DIABETES MELLITUS

Type 2 diabetes mellitus, is otherwise called as non – insulin dependent diabetes. The prevalence of the disease is still rising worldwide.

RISK FACTORS

Familial Aggregation

Risk is increased two- to five fold if a parent or sibling has the disease.

Genes

Genes responsible for T2DM is identified only in small number.T2DM is genetically complex disease. Putative susceptibility genes are identified by Genomic scans. Genomic scans identify the locus or loci which are in chromosome. Genes vary from population to population.

Obesity

Obesity is one of the components of T2DM. It is the most important predictor in disease process. In non obese persons, it is low in incidence.

Physical Activity

Many studies indicate the important role of physical activity in the development of T2DM. Reduced physical activity patients have high BMI, high BP.

GESTATIONAL DIABETES

In T2DM, gestational diabetes (diabetes first recognized during pregnancy) is the important risk factor in women.

OTHER RISK FACTORS

Reduced birth weight

Intrauterine diabetic environment

Exposure to metabolic environment.

Low Birth Weight

Reduction in Beta cell mass is due to nutritional deprivation. This is due to “thrifty phenotype”. It is an acquired defect expressed as T2DM.Low birth weight is also associated with systemic hypertension, insulin resistance and dyslipidemia.. There are certainly paternally derived genes that are linked to low birth weight.

Intrauterine Environment

The intrauterine environment increase the risk of T2DM.At early age itself, offspring of diabetic pregnancies develop T2DM.These patients

insulin secretary capacity is in reduced level. Diabetes development is 3 fold higher in patients those who are born to Diabetic mother. It is due to intrauterine programming.

Inflammation

Several markers are also associated with T2DM and its metabolic precursor, insulin resistance. Those are IL6,CRP,other cytokines, fibrinogen and plasma activator inhibitor – 1.The extent to which these proteins are involved in the pathogenesis of T2DM is not clear.

Sex Hormones

Reduced levels of sex hormones binding globulin leads to development of T2DM in females. Men with testosterone level and women with increased level of androgen are resistant to insulin. Insulin resistance, or hyperinsulinemia, and hyperandrogenemia and nulliparity are also characteristic of the polycystic ovarian syndrome.

REVIEW OF LITERATURE

In the body largest organ is skin. It can be easily inspected for scientific purposes. Physical signs of diabetes is due to dysregulation of glucose, insulin, and lipids. NEG of cutaneous proteins is due to chronically elevated blood sugar. This leads to advanced glycation end products (AGEs).Chronically elevated blood sugar results in nonenzymatic glycosylation (NEG) of cutaneous proteins ,which leads to irreversible advanced glycation end-products (AGEs).AGEs have been proposed as the mechanism for thickened skin and scleroderma-like changes and limited joint mobility. Structural proteins, such as collagen, that have undergone NEG become insoluble and resistant to degradation. Certain AGEs are related to glycemic control whereas others change with age as well as with glycemic control. In intensive insulin therapy for patients in DCCT results in lower levels of skin collagen AGEs, reduction in HbA1C and reduction in microvascular complications. Normal activities of insulin and lipoprotein lipase are necessary for normal production and catabolism of certain plasma lipids particularly VLDLs. Diabetes is most common cause of severe hypertriglyceridemia. Individuals with insulin resistance and insufficient levels of insulin have high levels of serum triglycerides with cutaneous xanthomas. On the other hand, individuals with insulin resistance and hyperinsulinemia have clinical manifestations of acanthosis nigricans. There

are also profound changes in cutaneous structure and function based on abnormalities of nerves and blood vessels. Studies document that cutaneous sensory innervation is diminished in diabetes compared with controls. Nerves and neuropeptides are important for normal immune function and normal tissue repair. Poor tissue repair and infections contribute to skin ulcers. It has been documented that non healing ulcers are the frequent cause of amputation.

Structural abnormalities of blood vessels include (1) increase in the overall thickness of the walls of the microvessels, (2) increased number of gaps between endothelial cells and pericytes in postcapillary venules, (3) homogenous basement membrane thickening between endothelial cells and pericytes of thermal micro vessels,(4) an increase in size and number of periadventitial fibroblast-like cells (veil cells).Functional abnormalities of the diabetic microvasculature include (1) increased permeability, increased baseline capillary pressure and decreased capillary peak blood flow following arterial occlusion, (2) normal resting blood flow to capillaries in dorsal fingers and toes, but a decreased response to heat induce hyperemia,(3) an increased resting cutaneous blood flow in the legs and the feet of diabetic patients with neuropathy but deficiency in the normal hyperemic response to heating. The defective hyperemic response was greater in the legs than arms and correlated with duration of diabetes,

retinopathy and proteinuria. Skin conditions that appear to be directly linked to endocrine, vascular, neurologic and immunologic impairment seen in diabetes mellitus. These conditions include ulcers, acanthosis nigricans, diabetic thick skin, cutaneous infections, cutaneous xanthomas. Number of the other clinical conditions pathobiology of disorders remains unclear. These include necrobiosis lipoidica, granuloma -annaulare, diabetic dermopathy, bullosis diabeticorum and acquired perforting dertomasis. Vasculopathy is a major factor in the pathogenesis of diabetic ulcers. The greatest risk factor for amputation in a study by Reiber and associates was low cutaneous blood flow as measured by transcutaneous oxygen pressure. There was a 16 fold excess risk of amputation if the TcPo2 was below 20 mm pg. The most important predictors of non healing were low TcPO2 and high TcCo2 in the skin adjacent to the ulcers.

Sensory neuropathy is also a major factor associated with diabetic ulcers and lower extremity amputation. Prevalence of symptomatic sensory neuropathy is 30 – 40% in diabetic patients compared to about 10% in the non diabetic population. Diabetic patients lacking vibratory sensation had a 15.5- fold excess risk of amputation compared to diabetic patients with intact vibratory sense. Clinical examination and the use of a 5.07 Semmes-Weinstein filament was the most sensitive test in identifying patients at risk for foot ulceration. Neuropathy and limited joint mobility was found to be

the most important etiologic factor for foot ulcers occurring on high pressure areas of patients with diabetes. Unperceived trauma such as blisters and ingrown toenails also relate in part to diminished influences of neuropeptides on skin immunity and tissue repair. Ulcers heal very slowly in diabetic patients with neuropathy.

Management of lower extremity ulcers requires if stasis dermatitis is present, it should be treated with topical steroids on the skin adjacent to the ulcers. Edema control is essential. Peripheral edema was a significant causal factor in the pathway leading to lower extremity ulceration.TcPo2 may improve significantly when leg edema is treated by bed rest, leg elevation, sodium restriction, or appropriately used diuretics. Unna boots, ache wraps, compression stockings, and even contact casting may be useful. If severe arterial insufficiency occurs it does not preclude these interventions. Treatment of underlying local soft tissue infection is important.

15% of diabetic patients with lower extremity ulcers develop osteomyelitis. Osteomyelitis should be suspected in deep or chronically draining ulcers, particularly if bone is exposed. Diabetic patients with suspected osteomyelitis from a foot ulcer might receive a 10 week course of culture guided antibiotics followed by surgical debridement. Mechanical protection for neuropathic extremities is another key element in therapy of diabetic ulcer.

Trials of growth- promoting factors and skin equivalents have been under investigation to enhance diabetic ulcers healing. New technologies for the topical treatment of diabetic treatment of diabetic foot ulcers show "modest benefit" if used with adequate off loading, debridement, and control of infection. LoGerfo and associates advocate the expanded role of arterial reconstruction for severe diabetic ulceration and gangrene, especially vein bypass graft to the dorsalis pedis artery. Prevention is by

1. Daily foot inspection, particularly for neuropathic patients. Advise careful selection of footwear that is not rigid or constricting in design or requiring a break-in period. Fitting shoes or socks were the most common reasons for foot ulcers. Advise patients to inspect shoes for foreign bodies before putting them on and to avoid walking barefoot.
2. Early health care attention for calluses, blisters, ingrown toe nails, dermatitis or athlete's foot.
3. Personally inspect the feet of diabetic patients at each visit.
4. Advice patients with a history of ulceration that they are at high risk for re ulceration (34% at 1 year, 61% at 3 years and 70% at 5 years.)

ACANTHOSIS NIGRICANS

Acanthosis Nigricans (NA) is a skin disorder that is a marker of hyperinsulinemia and insulin resistance. The clinical features include a velvety, warty hyperkeratosis which is black or grey brown in color,

observed on the back, and the sides of the neck. axillae, anogenital region and other skin fold. The neck is the most consistency affected area, and it is also the most reliable area for quantification of AN.

The Prevalence of type 2 diabetes mellitus in this subpopulation of Native Americans with AN is 50% in individuals above the age of 40. AN has also been shown to be a risk factor for development of T2DM. AN is strongly correlated with hyperinsulinemia. It has been demonstrated to be independent risk factor for the development of ischemic heart disease. AN used as an inexpensive surrogate marker for hyperinsulinemia to identify children and adolescents with a future risk of T2DM and ischemic cardiovascular disease. Mechanism for Acanthosis Nigricans (AN) is due to excess binding of insulin to insulin like growth factor receptors and androgens excess. DNA synthesis and cell proliferation in vitro through the IGF-1 receptors in fibroblast is by high concentration of insulin.

Certain drugs such as nicotinic acid and diethyl-stilbestrol have been reported to cause AN. Treatment for AN is generally ineffective, although disappearance of AN has been observed in obese individuals with weight reduction to IBW and with discontinuation of offending drugs.

DIABETIC THICK SKIN

Diabetes generally has thicker skin than their non diabetic counterparts. Collier and colleagues used ultrasound measurements to show that flexor forearm and medial upper arm skin in patients with diabetes

(mean age 25) was thicker than in controls. Diabetic patients with disease duration greater than 10 years and those with LJM of the hand had the greatest mean skin thickness.

SLS in children and young adults with type 1 diabetes mellitus is otherwise called as “diabetic hand syndrome”, limited joint mobility or “cherioarthropathy”. By palpation, thickening and induration of skin on dorsal fingers may be demonstrated. SLS and LJM typically begin on the fifth finger and progress radially, as well as from distal to proximal. With increasing duration of diabetes, the changes may be seen proximal to the metacarpophalangeal joint.

LJM is thought to result from deposition of connective tissue in the periarticular soft tissues around the joint capsule rather than being the result of a true arthropathy. Criteria have been defined for staging the severity of LJM and quantitating restrictions in motion with a goniometer.

Approximately 50% of adolescent patients with T1DM for more than 5 years are affected. Adults with T2DM may be affected as well. Patients with T1DM, correlation was found between LJM or SLS and duration of DM. LJM or SLS correlated with retinopathy. Other sign of micro vascular complications have been reported to be associated with LJM or SLS retinopathy appears to be the strongest association. In a recent cross –

sectional study, 83 patients with LJM and T2DM were compared with 56 diabetic patients without LJM for evidence of diabetic complications.

Cerebro vascular disease is 4 times common with LJM. Nephropathy is 3.3 times common. Coronary heart disease is 3 times common in patients of LJM. Longitudinal control of diabetes as measured by HbA1C was strongly associated with the presence or absence of LJM.

Pathogenesis of this condition is controversial evidence for the role of non enzymatic glycosylation of connective tissue is becoming stronger. Advanced glycation end products (AGEs) extracted from skin correlated significantly with signs of LJM in two studies. The most compelling data come from a longitudinal prospective study showing a two to three fold excess risk of LJM with HbA1C levels above 8% over a 2 year period. For every 1% increase in HbA1C level there was a 2.5 fold excess risk of LJM. Intensive insulin therapy may be beneficial in the treatment of LJM and SLS.

The reduction in skin AGEs suggests the effect of intensive insulin therapy may be beneficial. Physical therapy to preserve range of motion should be considered. Intensive insulin therapy in 122 patients in the DCCT resulted in lower levels of skin collagen AGEs.

Scleredema is usually described as a rare complication of Diabetes. In two studies the prevalence was reported to be 2.5% of 484

diabetic outpatients in a Veteran Administration Medical center in the United States and 14% of 100 hospital based diabetic patients at Kuwait University in Kuwait. Scleredema in diabetic patients is characterizes as firm non pitting edema of the skin, symmetrically distributed over the posterior neck, upper back and shoulders. It is asymptomatic, a fact that could lead to considerable under reporting of the phenomenon by both patients and physicians. A population based study would provide the best data regarding the true prevalence of scleredema of diabetes.

Scleredema of diabetes or scleredema diabeticorum is often reported as part of the spectrum of scleredema adultorum. It may be seen in both T1DM and T2DM, but much more common in type 2 disease. These type of scleredema are similar to the scleredema of diabetes in clinical appearance except they involve the face more often and may resolve spontaneously within 2 years. Monoclonalgammopathy and multiple myeloma have been reported with scleredema.

The pathogenesis is unclear. Ohta and collaborators showed that serum from patients with scleredema and a paraproteinemia caused enhanced synthesis of extracellular macromolecules in cultured fibroblasts. Case reports regarding the use of electron beam radiation, penicillin, cyclosporine, both PUVA and prostaglandin E1 have recently been referenced.

CUTANEOUS INFECTIONS

Modern metabolic management and antibiotic therapy, contributed to the view that the incidence of furunculosis, carbuncles, erysipelas and epidermophytosis is increased in diabetic patients. Infection clearly makes diabetes more difficult to manage and conversely hyperglycemia and ketoacidosis diminish chemotaxis, phagocytosis and bactericidal ability of white blood cells. It is difficult to correlate hyperglycemia with skin infections.

Both a retrospective and a prospective population based study shown diabetes is the commonest risk factor for invasive group B streptococcal infection in adults in both responsible and prospective population based study. Skin and soft tissues were the most common local sites of infection. Approximately 30% of the cases occured in patients with diabetes and overall case mortality rate was 21-32%.Diabetes is also associated with a 3-7 fold increased relative risk of invasive group A streptococcal infections.

MEO is a pyogenic infection of the ear canal with pseudomonas aeruginosa which is characteristically seen in older diabetic patients with purulent discharge, facial swelling, unrelenting pain, hearing loss and granulation tissue in the ear canal. The mortality rate is cited as 20-40% despite appropriate antibiotics.

Necrotizing fascitis is another uncommon but potentially lethal infection seen post operatively and following minor trauma as well as at injection sites. Organism may include facultative strepto-cocci, Bacteriodes, peptostreptococci and staphylococci. The perineum, trunk, abdomen and upper extremities are most commonly involved. Toxicity is often out of proportion to signs, Redness, induration, cyanosis and necrosis with overlying bullae may be seen. The most important aspects of treatment is early aggressive surgical debridement.An increased rate of cutaneous staphylococcal infection in a diabetic patients using continuous subcutaneous insulin infusion (CSII).Increase in the rate of staphylococcal carriage could not be demonstrated for patients using CSII compared with diabetic patients injecting insulin or normal control subjects.

Erythrasma is an infection of intertriginous skin with corynebacterium minutissimum. The organism produces a porphyrin pigment that results in characteristic coral red florescence when a wood's lamp is shined on the skin. Bacterial infections may be treated with appropriate systemic antibiotics. Erythrasma may be treated with topical or oral erythromycin.

Candida albicans causes angular cheilitis, glossitis, vulvovaginitis, balanitis, finger web space infection and paronychia in diabetic patients. Candida infection is common in poorly controlled diabetes. Clinical

evidence of candida paronychia was observed in 9.6% of 250 diabetic women compared with only 3.4% of 500 nondiabetic women. Candida infections may be treated with oral or topical anti fungals.

The prevalence of toenail onychomycosis appears to be increased in patients with diabetes. The importance of treating dermatophyte infections on the feet of diabetic patients is that they may provide entry for subsequent bacterial infections. Topical antifungals usually suffice to control fungal infection on skin adjacent to affected nails, but oral agents are needed to clear the onychomycosis.

Mucormycosis is an infection caused by a ubiquitous fungal organism, found primarily in soil and decaying vegetation. It has been repeatedly reported in diabetic patients. Rhizopus and Mucor have been observed complicating skin ulcers on legs and hands in diabetic patients. A destructive nasal mucosa and sinus infection called rhino cerebral mucormycosis (RCM) is particularly devastating. Facial or ocular pain and nasal stuffiness is the easy manifestation. Amphotericin B and surgical debridement are the treatment of choice. Mortality rates for RCM have been reported to be 15-34%.

ERUPTIVE XANTHOMAS

Diabetes is the most common cause of acquired hypertriglyceridemia. Triglyceride-rich chylomicrons are usually rapidly

cleared by the action of lipoproteins lipase. Lipoproteins lipase activity is decreased in uncontrolled diabetes. Insulin is necessary for normal clearance of plasma lipoproteins. Eruptive xanthomos may develop as the skin manifestations of chylomicrons in hypertriglyceridemic patients. Plasma lipoproteins can be shown to enter the skin, where they are phagocytosed by macrophages. These macrophages appear as foam cells in the eruptive xanthomas.

Eruptive xanthomas appear as 1 to 4 mm size most commonly present in extensor surface of arms, legs and buttocks. They appear as reddish yellow papules. They are usually asymptomatic, but these are the most important clinical signs because they may be the first indication of diabetes. The hypertriglyceridemia responds rapidly to diet and insulin therapy and the eruptive xanthomas usually resolve completely in 6-8 weeks.

NECROBIOSIS LIPOIDICA (NL)

The most recognized skin condition associated with diabetes is its taxonomy. The term necrobiosis lipoidica diabeticorum in 1932 generation of dermatologists and diabetologists have used the abbreviation “NLD” to refer to this condition. If one adds the term “diabeticorum” to search about 80% of the published.

NL was associated with diabetes in approximately two-thirds of 171 cases reported from the Mayo clinic. Glucose tolerance tests performed on non diabetic patients revealed another 5-10% with abnormalities in carbohydrate metabolism. It is seen on scalp, fingers, hands, forearms, face, and nipple. It may be solitary.

The clinical presentation is usually the key to diagnosis but the histology is also characteristic. It is difficult to distinguish NL from other necrobiotic condition such as granuloma annulare. The histopathologic and immuno histochemical features of NL have been succinctly reviewed and contrasted with granuloma annulare (GA).The histological features of NL in middle to deep dermis are histiocytic granulomas with necrobiosis and periodic acid Schiff (PAS) positive staining. Nerve staining with s-100 is diminished in lesional skin and in the inflammatory perilesional areas.

The pathogenesis of NL remains unknown but many mechanisms have been proposed and reviewed. Proposed mechanism include hereditary, microangiopathy, increased production of fibronectin by endothelial cells, increased factor VIII related antigen, abnormal platelet function and prostaglandin synthesis, accelerated collagen aging and immune –mediated vasculopathy.

NL and GA are characterized by altered extracellular matrix and degenerative changes in collagen and elastic fibers. There is no evidence

that poor glycemic control is a factor in NL although this has not been extensively studied using newer techniques for subfractionating AGEs from skin biopsis.TcPo2 value are significantly lower in lesional skin than in adjacent normal skin.

The long list of treatments for NL is evidence that no treatment is the accepted standard of effective care. Experience with dipyridamol and aspirin has been disappointing. Treatment for early inflammatory phase of NL is high potency topical steroids. Perilesional skin is injected by injection triamcinolone, ulceration may occur with local steroids. Treatment is short term use of systemic steroids and pentoxifylline 400 mg three times a day.

The main issues for plaque type NL are the cosmetic appearance and protection to avoid ulceration. Cover-up products such as Derma blend or cover mark may be useful for special occasions to help cosmetically Protective padding is well advised for ulcer protection during high risk activities and a night light is useful to prevent inadvertent trauma when using the bathroom at night.

When ulcers occur in NL, the same principles apply as discussed above for diabetic ulcers. A report of the successful use of cyclosporine for the treatment of persistently ulcerated NL in two patients was published. Very large or recalcitrant ulcers in NL excision done. Split thickness skin grafting appears to be effective and the preferred therapy.

GRANULOMA ANNULARE (GA)

GA is a benign self-limited condition characterized by annular plaques usually seen over the dorsal hands, feet or ankles. The plaques often consist of a ring of papules, red to reddish purple in color, with clearing or flattening in the center. They may be solitary papules, nodules or plaques and on rare occasions presents as subcutaneous nodules or perforating lesions. About 15% of patients have more than 10 lesions and 7-10% has generalized lesions. The localized form tends to come on early in life and usually clears within 2 years, whereas in the generalized form the mean age of onset is 52 years and it rarely clears spontaneously.

The pathogenesis of GA remains unknown. Its association with diabetes has long been debated. Diabetes is present in 9.7% of 1353 patients with localized lesions of GA and 21% of 100 patients with generalized GA. In two more recent retrospective studies 11 of 61 patients (18%) and 10 0f 84 patients (12%) with GA has diabetes. A large population based study of patients with DM, but the more recent data continue to support the arguments in favour of an association.

Treatment with high potency topical steroids or steroids injection may be effective for localized GA, but it is usually an asymptomatic self-limited process.GA may be more pruritic and persistent and it is certainly more cosmetically disabling. Encouraging results for generalized GA have been

reported with PUVA. The sunburn has been reported as a precipitating factor for some patients with generalized GA. Limited success has been reported with systemic corticosteroids, chloroquine, potassium iodide, sulfones, niacinamide and chloropropamide as reviewed. GA lesions have been reported in patients with HIV infection. If risk factors for HIV are present antibody testing should be considered.

DIABETIC DERMOPATHY

Melin is generally credited with describing atrophic circumscribed lesions localized to the lower extremities in diabetic patients. They consist of small (2-10mm), rounded, brownish, atrophic lesions almost exclusively over the pretibial surface of the legs. They have subsequently been referred to most commonly as “shin spots” and diabetic dermopathy. Controversy has surrounded this condition regarding the prevalence, the specificity as a cutaneous marker for diabetes, the relationship to other complication of diabetes and its relationship to trauma.

Prevalence of atrophic shin spots ascertained from diabetic patients in outpatient clinics has ranged from 24-65% for males and 4-39% for females. The prevalence in non diabetic control groups was 7% of 104 patients in a hypertension clinic, 20% of 183 patients in an endocrine clinic, 3% of 100 patients in dermatology clinic and 1.6% of 201 healthy medical students.T1DM and 39% of patients with T2DM compared with 2% of a

control group comprised of 100 healthy people, mainly hospital personnel aged 15-50 years. Diabetic dermopathy does occur with greater frequency in patients with diabetes. There is an agreement that it occurs more frequently in men and the duration of diabetes is directly related to the prevalence of diabetic dermopathy.

The pathogenesis of this condition is unknown. Trauma has been the leading candidate even though most patients are unaware of associated trauma and the lesions are asymptomatic. Arguments in favor of trauma are supported by the location on the shins and increased prevalence in men. Melin was unable to produce lesions on the shins of diabetic patients with a rubber hammer and found no lesions on the shins of normal health soccer players. Linther was able to induce atrophic circumscribed skin lesions at the site of trauma from locally applied heat and cold in patients with diabetes. Diabetic dermopathy represents a pattern of repair in response to injury or inflammation within the milieu of diabetes.

Melin found an association of atrophic shin spots with other clinical complications of diabetes, including retinopathy, lower extremity vascular disease and nephropathy. Patients with DM a strong statistical relationship with retinopathy, nephropathy and neuropathy was reported. The histology is not diagnostic. It shows epidermal atrophy and pigment inclusion but no clear cut evidence for a vasculopathy in lesional skin by either histology or

electron microscopy. No treatment is necessary since it is without symptoms or morbidity.

BULLOSIS DIABETICORUM

The abrupt onset of bullae on the extremities has been reported as a rare condition associated with diabetes. The lesions are usually on the toes, feet and distal lower extremities but also observed on the fingers, hands and forearms. They are unrelated to any apparent trauma or infection and heal without scarring in 2-5 weeks unless they become infected. The condition may resolve spontaneously it may recur over a number of years.

The histopathology is variable as reviewed by Toonstra. The level of separation resulting in the bulla may be intra epidermal or sub epidermal. The intra epidermal split may occur anywhere from supra basally to sub corneally, usually without acantholysis. Spongiosis may be present. Direct immuno florescence is usually negative.

The pathogenesis is unknown and the treatment is supportive wound care. The main significance of this condition is recognition. The differential diagnosis includes bullosis impetigo, bullous pemphigoid, pemphigus vulgaris, epidermolysis bullosa acquista, porphyria cutanea tarda (PCT), bullous erythema multiforme and insects bite reaction. A negative culture for staphylococci and negative direct immuno florescence on skin biopsy and compatible histopathology makes this diagnosis by exclusion. A

porphyrin screen could be considered if other signs of PCT are present. The diagnosis is important because the treatment of a number of the other blistering disease involves the use of systemic steroids or immunosuppressive therapy which confers significant risk of toxicity to diabetic patients.

ACQUIRED PERFORATING DERMATOSIS

APD has been reported in association with diabetes and renal failure. Rapini and collaborators reviewed a variety of terms used for this group of conditions. They include Kyrli's disease, relative perforating collagenosis (RPC), perforating folliculitis and ellastosis perforans serpiginosa. Histology may vary from location to location on the same patient and the pathogenesis is unclear. It appears more logical to consider these conditions together rather than to emphasize differences.

APD is characterized by trans epidermal elimination of what appears to be altered collagen. The clinical appearance consists of pruritic papules and nodules on the upper and lower extremities as well as the trunk and to a lesser extent the face. Diabetes with at least one complication (nephropathy, retinopathy, peripheral vascular disease or cardio myopathy), 15 patients had nephropathy and 10 were on dialysis. Dialysis patients received prospective skin examination, 11% (8 of 72 patients) were observed to have APD. Seven of the eight patients had DM.

These observations are supportive of the APD with DM and chronic renal failure, and it suggests that APD may be more common than previously appreciated. The pathogenesis is unclear, but treatment with glycemic control, ultraviolet light, retinoic acid or topical steroids may be effective.

GLUCAGONOMA

Glucagonoma is one cause for diabetes that is potentially curable by surgery. This diagnosis should be considered in diabetic patients with calcitrant erosive dermatitis typically involving the central face, groin, friction areas and the distal extremities. It is usually seen in the clinical setting of weight loss, diarrhea, glossitis, anemia, hypo albuminemia and mild DM. Skin biopsy shows epidermal necrosis, edema and split in the upper spinous layer along with a neutrophilic infiltrate. The clinical and histologic features taken together resulted in the term necrolytic migratory erythema (NME).

Glucagonoma is a tumor of the alpha cells of the pancreas, which secrete glucagon. It is malignant in 60-80% of cases and often is metastatic at the time of diagnosis. The diagnosis is made more difficult by the fact that 15% of patients with glucagonoma do not have DM and some patients with NME have normal glucagon levels and no evidence of glucagonoma.

The tumors can usually be visualized on CT scan. Selective celiac axis arteriography is the most useful diagnostic study for localizing the tumors and identifying metastases in the liver. Treatment usually includes surgical excision. Even in metastatic disease, cytoreductive surgery often results in long remission. Chemotheraphy may be helpful and long-acting somatostatin analogues results in good symptomatic relief even though they have no effect on tumor size.

SKIN COMPLICATIONS DUE TO INSULIN INJECTION

Intensive insulin therapy effectively delays the onset and slows the progression of micro vascular disease in T1DM. External insulin pumps are an effective alternative for intensive insulin therapy. Management of the skin complications of insulin therapy may become more problematic with intensive insulin therapy.

Lipoatrophy with less pure earlier insulin preparations was reported in 16% of patients, with highly purified insulin it occurred in 0-2.5% of cases. Lipohypertrophy has been attributed to local immune complex formation and complement fixation with release of lysosomal enzymes in response to a presumed antigenic component of the less purified insulin. CSII with regular HI and speculated that it is responsible for the improvement.

Lipohypertrophy has not decreased with the use of highly purified insulin and HI. Lipohypertrophy was observed in 14% of patients using conventional insulin, 22% of patients injecting highly purified human insulin. It is thought that it occurs because of a local anabolic effect of insulin that promotes fat and proteins synthesis. The problem can usually be alleviated by rotation of injection sites but the hypertrophic sites are often repeatedly used by the patients because they tend to be less painful. Patients should be discouraged from using the sites with lipohypertrophy because the insulin absorption can be delayed or inconsistent.

Local and systemic allergic reaction has been reported with insulin injection. A variety of factors may be involved including contaminating proteins, zinc and protamine as well as insulin itself. Such allergic reaction are usually IgE mediated although patients may have IgE antibodies to insulin without clinical evidence of an adverse reaction. IgE antibodies have been measured in sera from allergic non diabetic individuals with affinities and concentrations similar to those seen in diabetic patients. Local reactions to insulin are often transient and no major significance. They usually presents as a wheal and flare within 30 minutes. Systemic allergic reaction though rare they are potentially life threatening. They include urticarial, angioedema, anaphylaxis and the Arthus reaction. Highly purified insulin and HI are less immunogenic, allergic reactions are still observed. Intra

dermal testing may be used to find an insulin preparation to which the patients does not react. Desensitization techniques are useful for generalized insulin allergies. Systemic steroids and antihistamines are occasionally required.

Non immunologic cutaneous reactions have been reported to insulin injection as well. They include infections, zinc granulomas, sterile abscesses, silicon oil from disposable syringes and keloid formation as well as pigmentation that resemble acanthosis nigricans.

Continuous subcutaneous insulin infusion (CSII) deserves special mention because skin complications are the most common reason for the treatment discontinuation. Chatelau and collaborators reported on 116 patients 14 cases of skin inflammation or infection during the study period.51% developed redness at the catheter sites, 19% developed subcutaneous nodules and 48% had whitish scars secondary to previous infections.

AIM OF STUDY

To study the various Mucocutaneous Manifestations of type 1 and type 2 Diabetes Mellitus

To study its relation with glycemic status, age and duration of diabetes.

STUDY PROTOCOL

PLACE OF STUDY:

Dermatology OPD, Dermatology ward, Diabetology OPD, medicine OPD and medicine ward in Stanley Medical College.

DURATION:

March 2014 to September 2014.

STUDY DESIGN:

Cross Sectional Study design.

SAMPLE SIZE:

250 Patients with diabetes mellitus (type 1 and type 2).

INCLUSION CRITERIA:

Patients with type 1 and type 2 Diabetes mellitus attending diabetology, dermatology and medicine OPD.

EXCLUSION CRITERIA:

HIV

CKD

Malignancy

Known case of primary skin disorders

On immune suppressive drugs

Pregnant and lactating women

MATERIALS AND METHODS

Diabetic patients those who are attending Diabetology OPD, Dertmatology OPD, medicine OPD will be screened for dermatological manifestation. Patients glycemic status is evaluated with FBS, PPBS. Dermatologist opinion will be obtained. If needed, to confirm this pathological evaluation and further investigation will be done according to dermatologist advice. Patients mucocutaneous manifestation will be treated according to guidelines.

ANALYSIS PLAN:

Data will be analysed with the SPSS software version 16.0for windows. All continuous data will be expressed as mean and SD and will be analysed using students test. Categorical data will be expressed as number (percentage) and analysed by chi square test.

OBSERVATION AND RESULTS

Study Groups

Treatment Groups	Name of Group	Study	Number of Subjects
Group A	Good Control	Mucocutaneous manifestation in diabetic study subjects with good blood glucose control(≤ 180 mg/dl)	26
Group B	Poor Control	Mucocutaneous manifestation in diabetic study subjects with poor blood glucose control(> 180 mg/dl)	224

Statistics

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with Unpaired t test and categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using EpiInfo software (7.1.0.6 version; Center for disease control, USA) and Microsoft Excel 2010.

Sample Size Calculation

Sample size was determined on the basis of a pilot study in which the prevalence of cutaneous lesions in diabetics was measured at 20%. We calculated a minimum sample size of 246 patients was required, assuming a type 1 error (two-tailed) of 0.05 and a margin of error of 10%. Therefore, the final sample selected was n=250.

$$n = \frac{t^2 \times p(1-p)}{m^2}$$

Description:

n = required sample size

t = confidence level at 95% (standard value of 1.96)

p = estimated prevalence of malnutrition in the project area

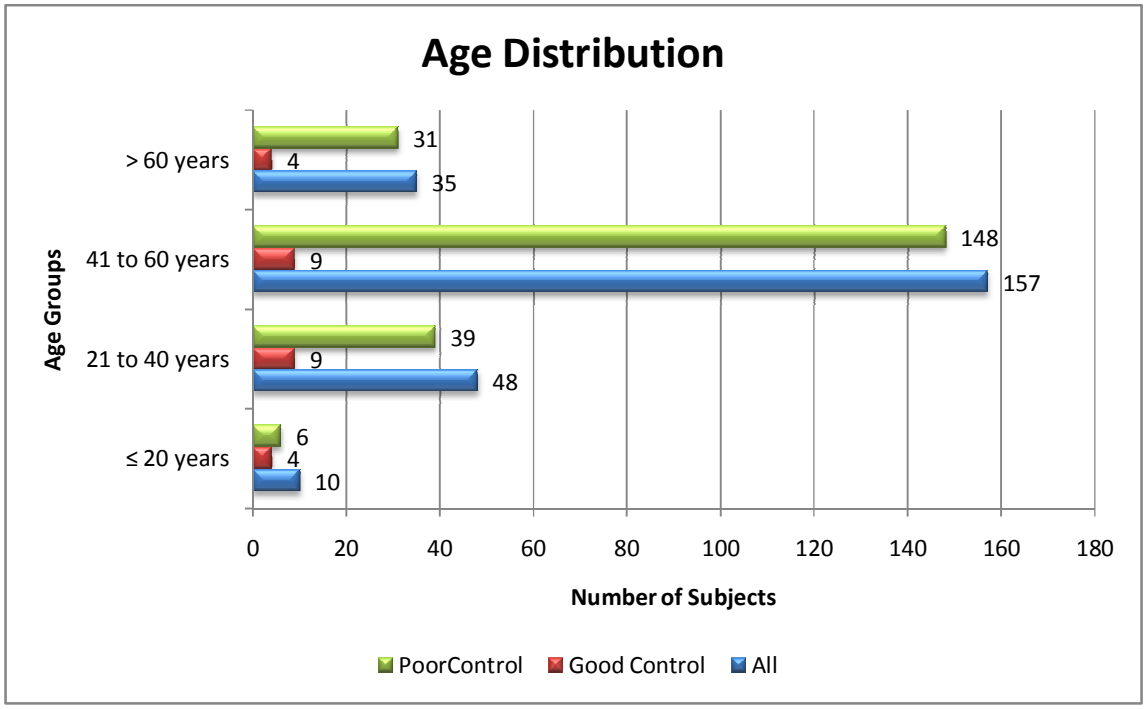
m = margin of error at 10% (standard value of 0.05)

$$n = \frac{(1.96)^2 \times 0.2(1-0.8)}{(0.05)^2}$$

$$n = \frac{3.8146 \times 0.16}{0.0025}$$

$$= 246$$

Age

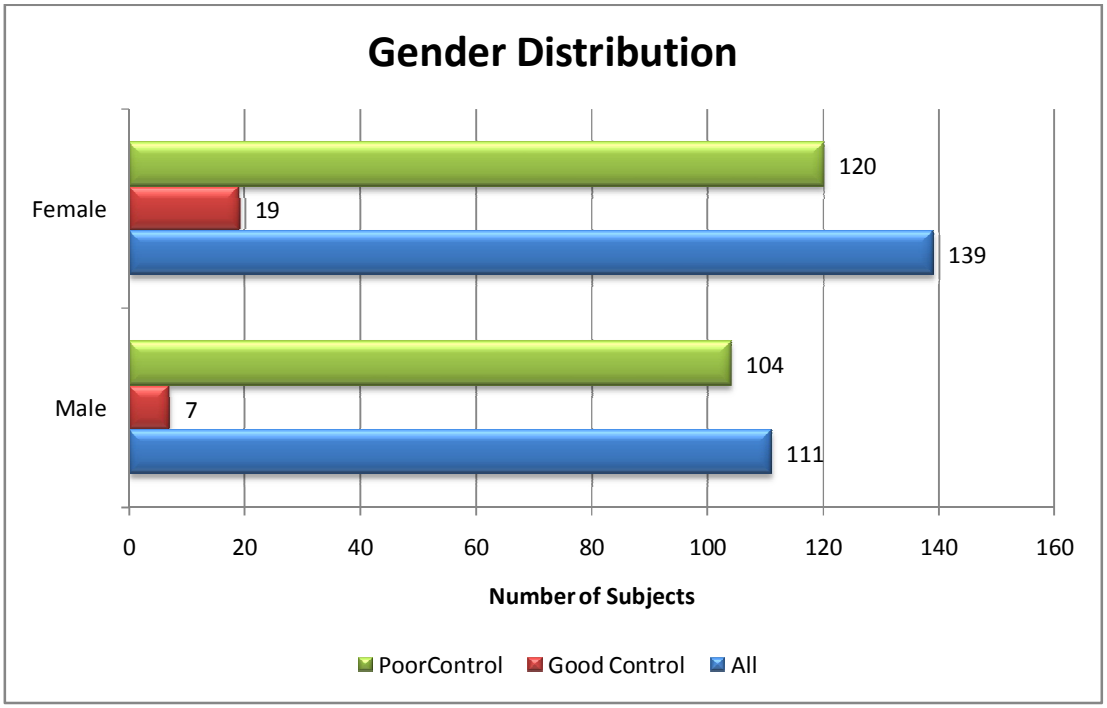


Age Distribution	All	%	Good Control	%	PoorControl	%
≤ 20 years	10	4	4	15.38	6	2.68
21 to 40 years	48	19.2	9	34.62	39	17.41
41 to 60 years	157	62.8	9	34.62	148	66.07
> 60 years	35	14	4	15.38	31	13.84
Total	250	100	26	100	224	100

Age Distribution	All	Good Control	PoorControl
N	250	26	224
Mean	48.376	42.19231	49.09375
SD	12.0165	17.08337	11.11478
P value Unpaired t test		0.54205296	

By conventional criteria the association between the study groups and age is considered to be not statistically significant since $p > 0.05$

Gender

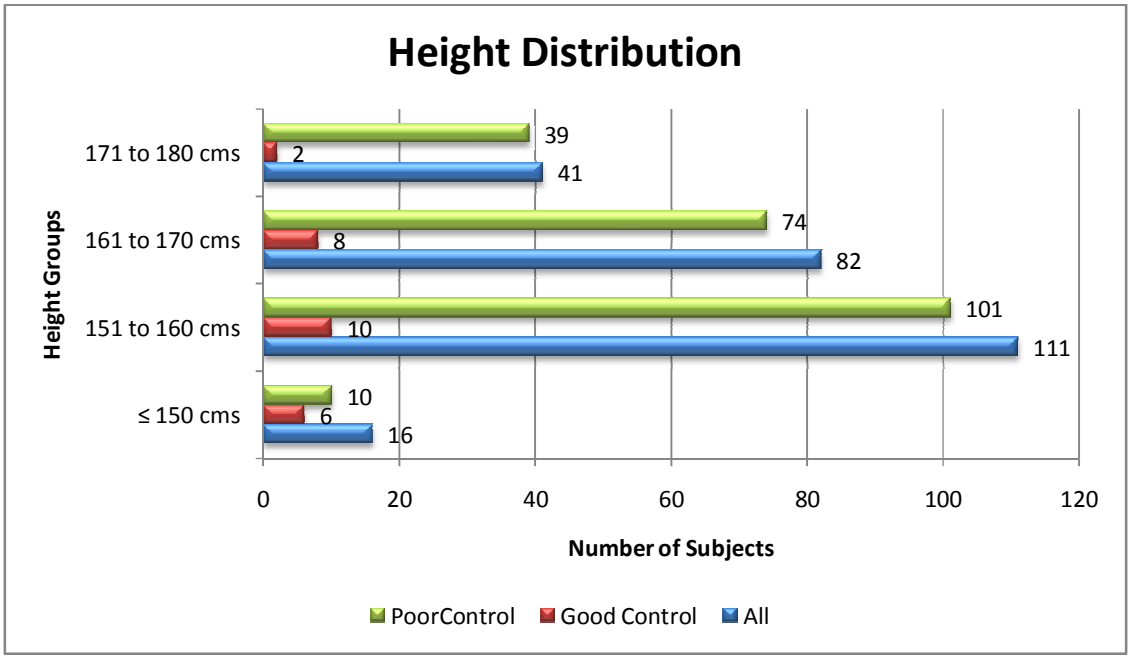


Gender Distribution	All	%	Good Control	%	Poor Control	%
Male	111	44.4	7	26.92	104	46.43
Female	139	55.6	19	73.08	120	53.57
Total	250	100	26	100	224	100
P value Chi squared Test		0.58116				

By conventional criteria the association between the study groups and gender is considered to be not statistically significant since $p > 0.05$.

Since age and gender is not statistically significant, it means that there is no difference between the groups. In other words the groups contain subjects with the same basic demographic characteristics.

Height

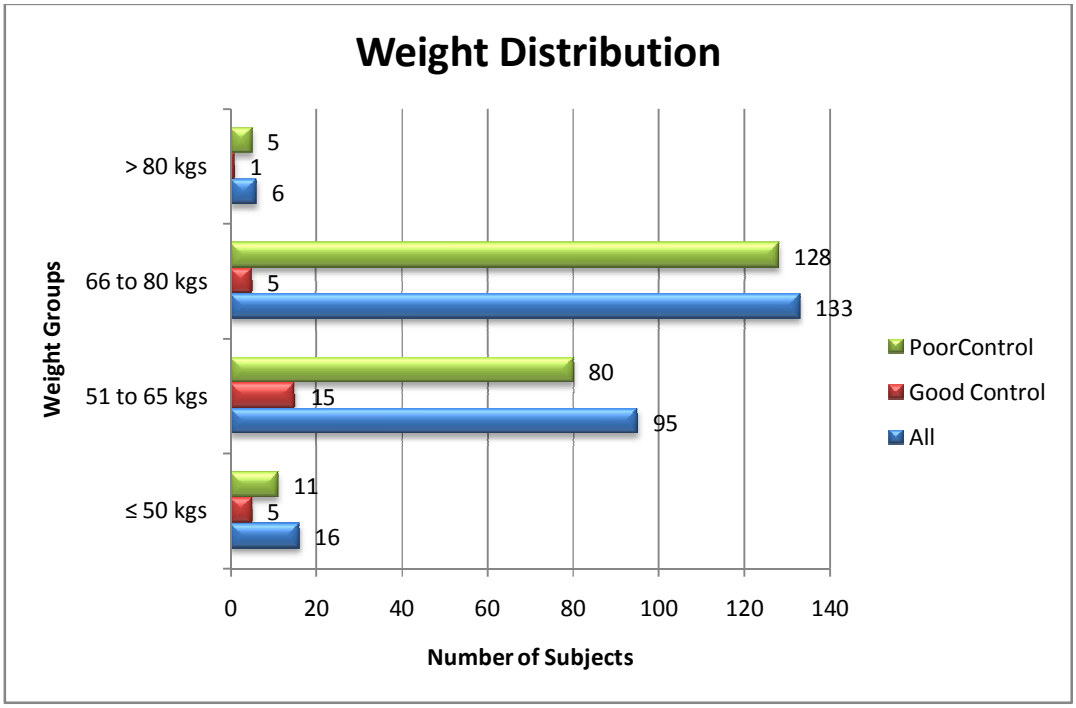


Height Distribution	All	%	Good Control	%	PoorControl	%
≤ 150 cms	16	6.4	6	23.07692	10	4.464286
151 to 160 cms	111	44.4	10	38.46154	101	45.08929
161 to 170 cms	82	32.8	8	30.76923	74	33.03571
171 to 180 cms	41	16.4	2	7.692308	39	17.41071
Total	250	100	26	100	224	100

Height Distribution	All	Good Control	PoorControl
N	250	26	224
Mean	162.012	158.2308	162.4509
SD	8.221083	9.052327	8.026151
P value Unpaired t test		0.30245622	

By conventional criteria the association between the study groups and height is considered to be not statistically significant since $p > 0.05$.

Weight

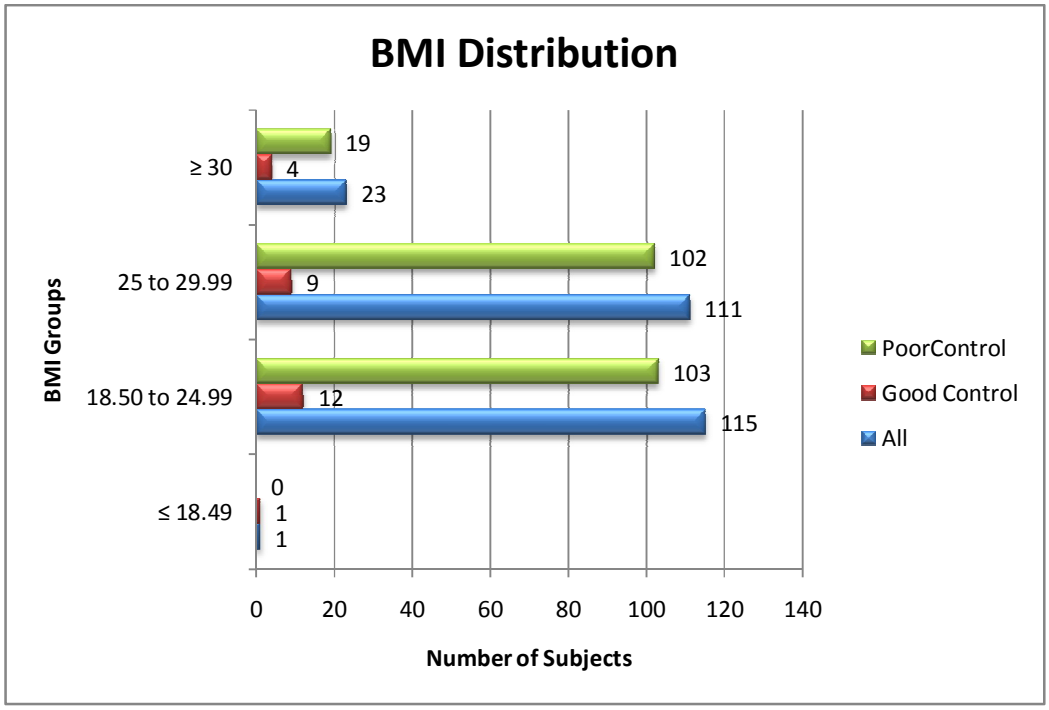


Weight Distribution	All	%	Good Control	%	PoorControl	%
≤ 50 kgs	16	6.4	5	19.23	11	4.91
51 to 65 kgs	95	38	15	57.69	80	35.71
66 to 80 kgs	133	53.2	5	19.23	128	57.14
> 80 kgs	6	2.4	1	3.85	5	2.23
Total	250	100	26	100	224	100

Weight Distribution	All	Good Control	PoorControl
N	250	26	224
Mean	66.7	61.61538	67.29018
SD	9.445787	9.670892	9.261555
P value Unpaired t test		0.7864537	

By conventional criteria the association between the study groups and weight is considered to be not statistically significant since $p > 0.05$.

BMI

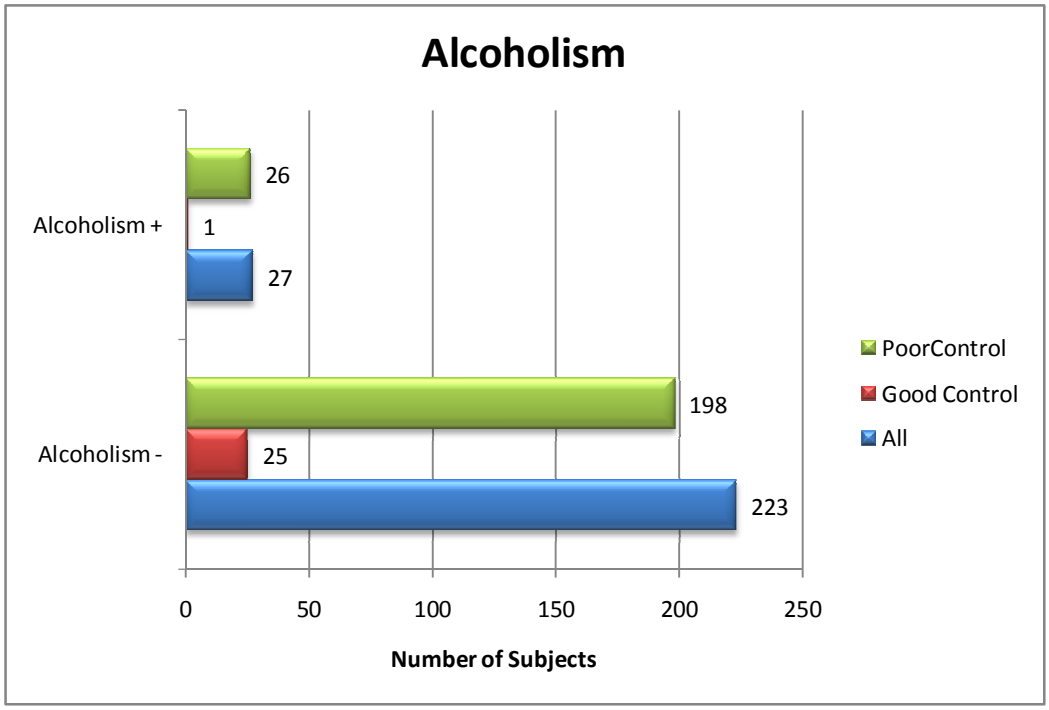


BMI Distribution	All	%	Good Control	%	PoorControl	%
≤ 18.49	1	0.4	1	3.846154	0	0
18.50 to 24.99	115	46	12	46.15385	103	45.98214
25 to 29.99	111	44.4	9	34.61538	102	45.53571
≥ 30	23	9.2	4	15.38462	19	8.482143
Total	250	100	26	100	224	100

BMI Distribution	All	Good Control	PoorControl
N	250	26	224
Mean	25.44934	24.95343	25.50691
SD	3.145144	4.14922	3.013834
P value Unpaired t test		0.514461049	

By conventional criteria the association between the study groups and BMI is considered to be not statistically significant since $p > 0.05$.

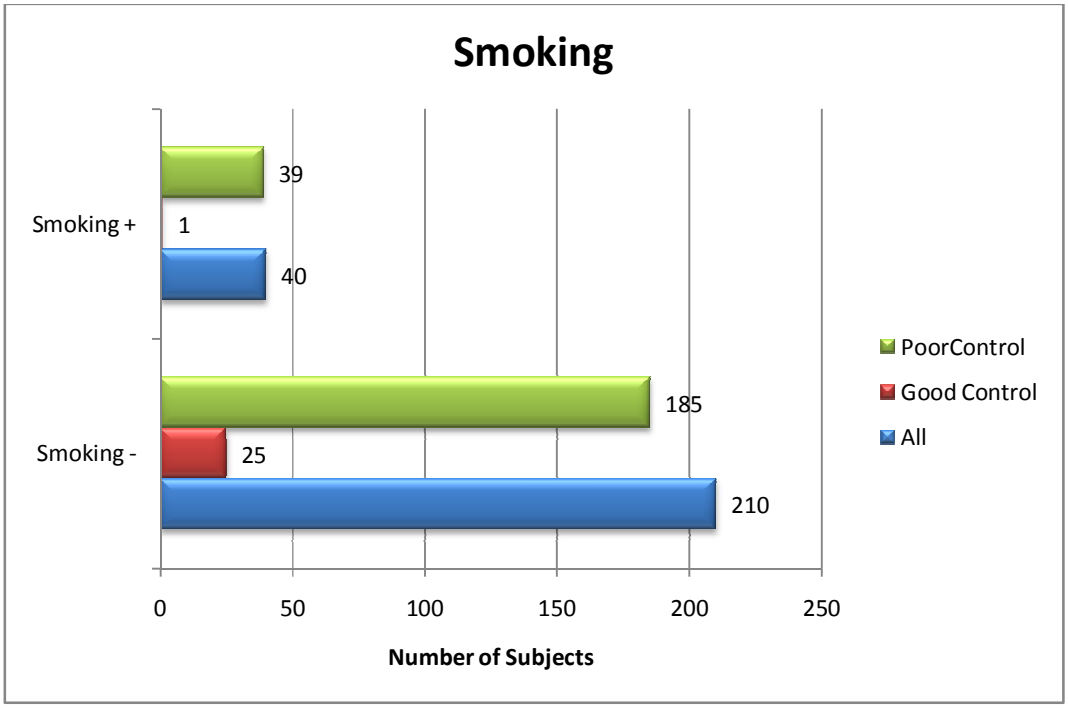
Alcoholism



Alcoholism	All	%	Good Control	%	Poor Control	%
Alcoholism -	223	89.2	25	96.15385	198	88.39286
Alcoholism +	27	10.8	1	3.846154	26	11.60714
Total	250	100	26	100	224	100
P value Chi squared Test		0.227479				

By conventional criteria the association between the study groups and alcoholism is considered to be not statistically significant since $p > 0.05$.

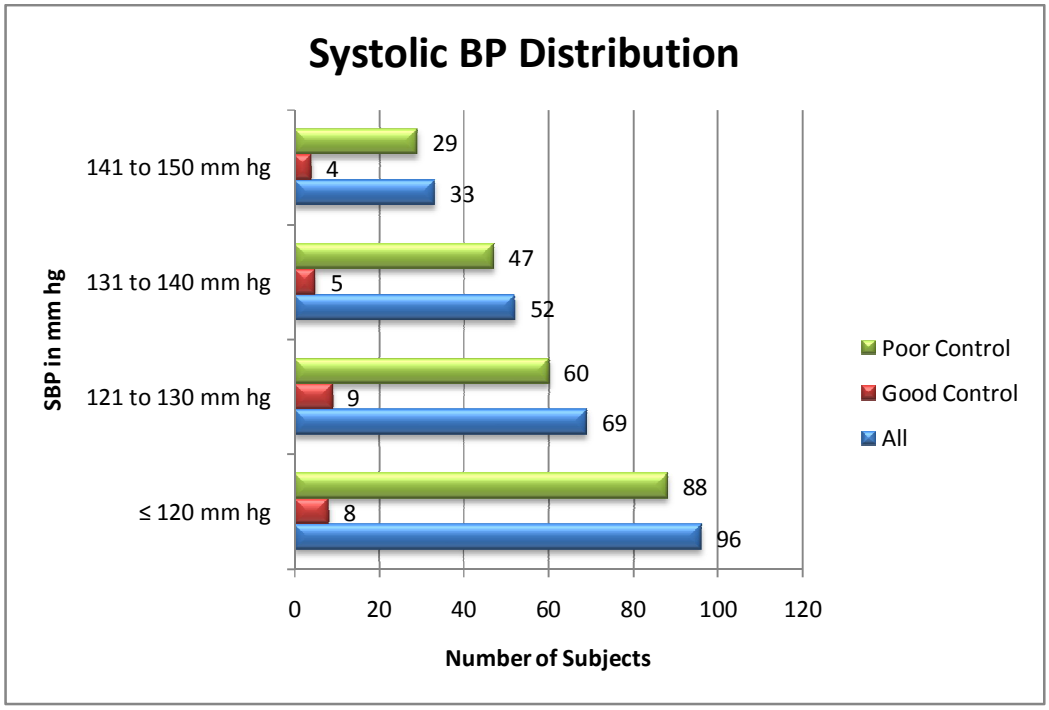
Smoking



Alcoholism	All	%	Good Control	%	Poor Control	%
Smoking -	210	84	25	96.15385	185	82.58929
Smoking +	40	16	1	3.846154	39	17.41071
Total	250	100	26	100	224	100
P value Chi squared Test	0.074122					

By conventional criteria the association between the study groups and smoking is considered to be not statistically significant since $p > 0.05$.

Systolic BP

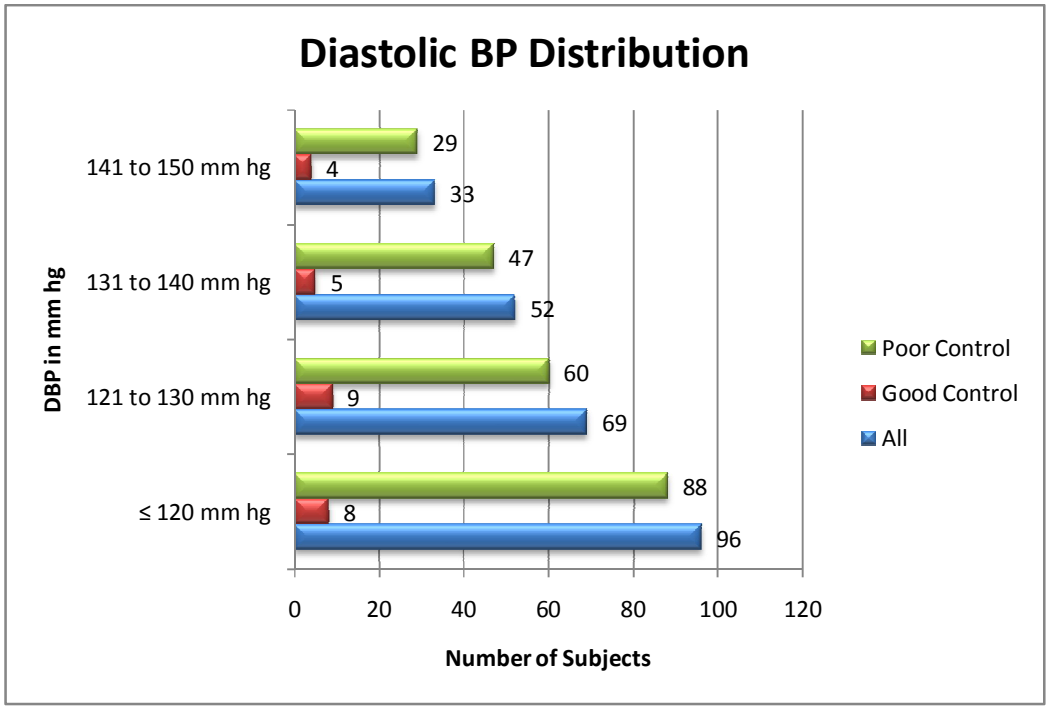


Systolic BP Distribution	All	%	Good Control	%	PoorControl	%
≤ 120 mm hg	96	38.4	8	30.76923	88	39.28571
121 to 130 mm hg	69	27.6	9	34.61538	60	26.78571
131 to 140 mm hg	52	20.8	5	19.23077	47	20.98214
141 to 150 mm hg	33	13.2	4	15.38462	29	12.94643
Total	250	100	26	100	224	100

Systolic BP Distribution	All	Good Control	Poor Control
N	250	26	224
Mean	130.064	130.3846	130.0268
SD	11.29221	11.55189	11.28749
P value Unpaired t test		0.88185124	

By conventional criteria the association between the study groups and systolic BP is considered to be not statistically significant since $p > 0.05$.

Diastolic BP

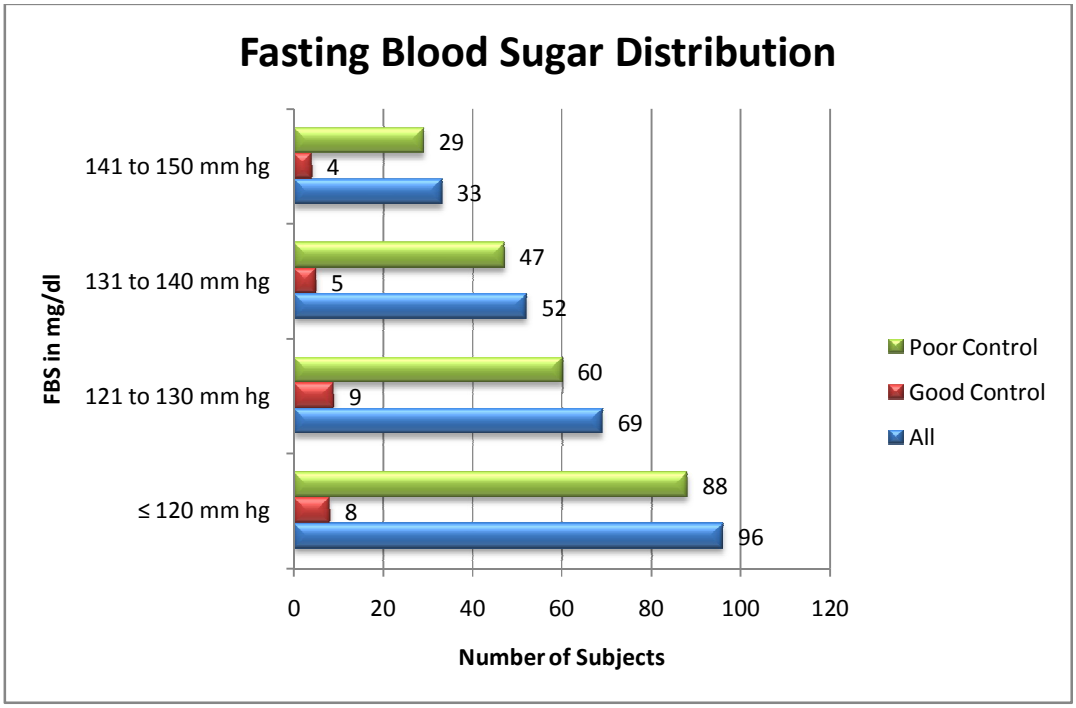


Diastolic BP Distribution	All	%	Good Control	%	PoorControl	%
≤ 70 mm hg	51	20.4	14	53.84615	37	16.51786
71 to 80 mm hg	129	51.6	6	23.07692	123	54.91071
81 to 90 mm hg	58	23.2	6	23.07692	52	23.21429
91 to 100 mm hg	12	4.8	0	0	12	5.357143
Total	250	100	26	100	224	100

Diastolic BP Distribution	All	Good Control	Poor Control
N	250	26	224
Mean	80.84	76.53846	81.33929
SD	8.550253	8.918434	8.384861
P value Unpaired t test		0.13797185	

By conventional criteria the association between the study groups and diastolic is considered to be not statistically significant since $p > 0.05$.

FBS



Fasting BS Distribution	All	%	Good Control	%	PoorControl	%
≤ 80 mg/dl	0	0	0	0	0	0
81 to 110 mg/dl	18	7.2	17	65.38462	1	0.446429
111 to 140 mg/dl	68	27.2	8	30.76923	60	26.78571
> 140 mg/dl	164	65.6	1	3.846154	163	72.76786
Total	250	100	26	100	224	100

Fasting BS Distribution	All	Good Control	Poor Control
N	250	26	224
Mean	161.612	107.0769	167.942
SD	42.87534	15.35428	40.48696
P value Unpaired t test		0.0000	

By conventional criteria the association between the study groups and fasting blood sugar levels among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the mucocutaneous lesions among diabetic patients, the fasting blood sugar levels in good blood glucose control group is predominantly less(107.07±15.35 mg/dl) when compared to poor blood glucose control group(167.94±40.49). It is statistically significant with a p-value of 0.0000 according to unpaired t-test.

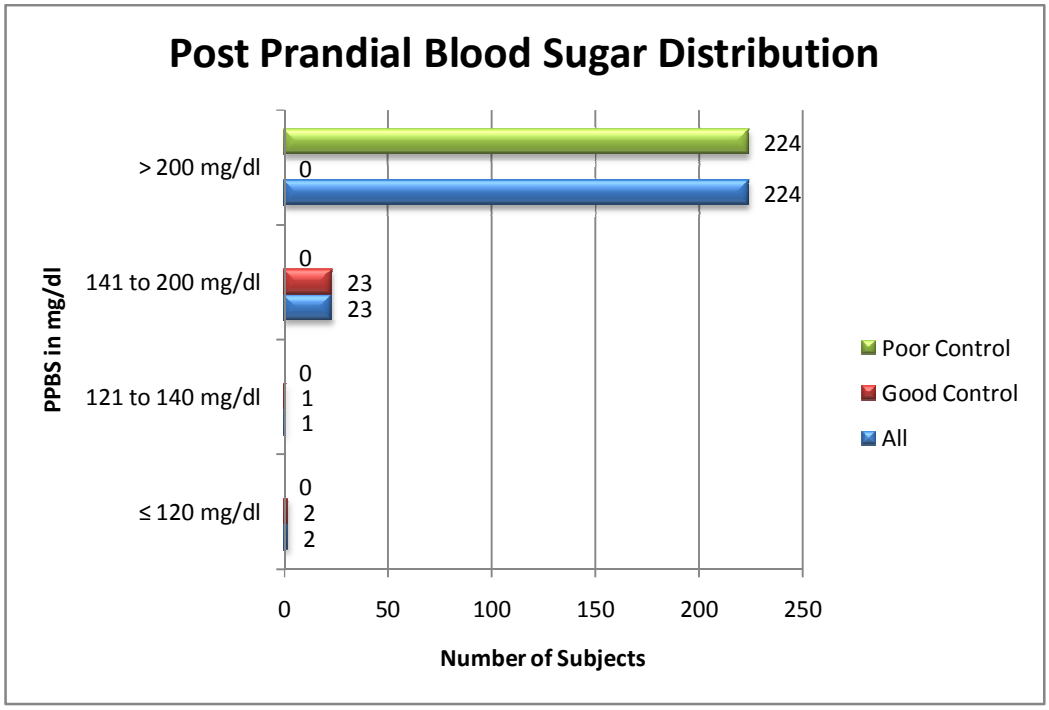
Clinical Significance

The average fasting blood sugar levels in good blood glucose control group is meaningfully less than poor blood glucose control by 57% with a mean difference of 60.86 mg/dl between the groups. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real reduction in fasting blood glucose levels among good blood glucose control group

PPBS



Post Prandial BS Distribution	All	%	Good Control	%	PoorControl	%
≤ 120 mg/dl	2	0.8	2	7.692308	0	0
121 to 140 mg/dl	1	0.4	1	3.846154	0	0
141 to 200 mg/dl	23	9.2	23	88.46154	0	0
> 200 mg/dl	224	89.6	0	0	224	100
Total	250	100	26	100	224	100

Post Prandial BS Distribution	All	Good Control	Poor Control
N	250	26	224
Mean	270.548	178.6538	281.2143
SD	52.55068	28.0855	43.54626
P value Unpaired t test		0.0000	

By conventional criteria the association between the study groups and post prandial blood sugar levels among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the mucocutaneous lesions among diabetic patients, the post prandial blood sugar levels in good blood glucose control group is predominantly less(179.65±28.08 mg/dl) when compared to poor blood glucose control group(281.21±43.55). It is statistically significant with a p-value of 0.0000 according to unpaired t-test.

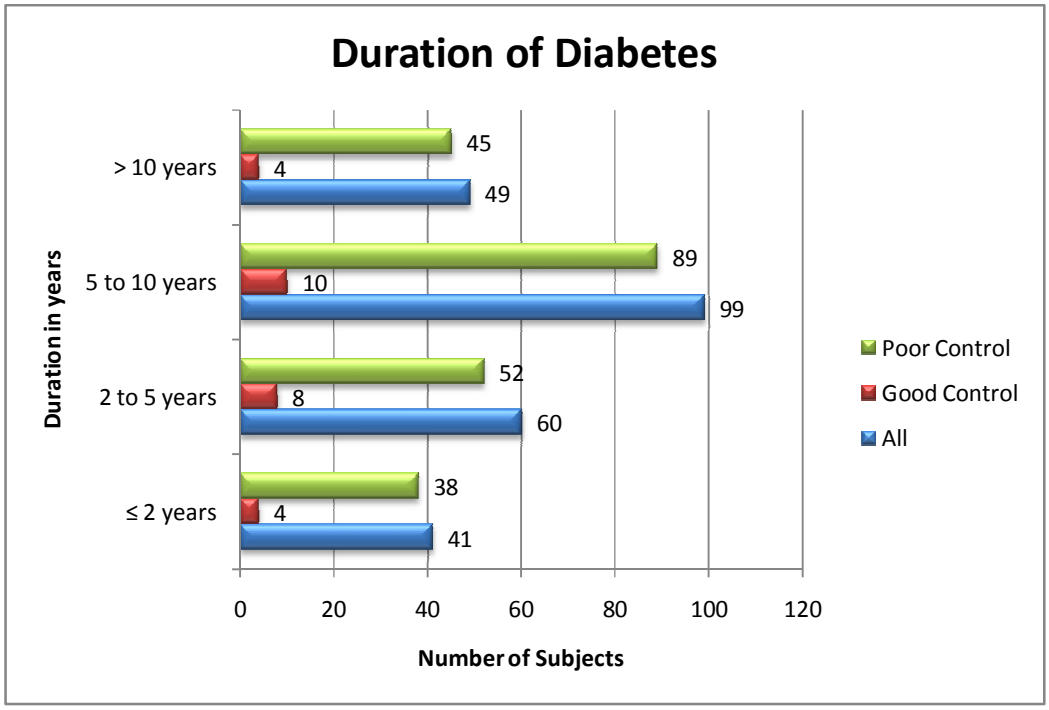
Clinical Significance

The average post prandial blood sugar levels in good blood glucose control group is meaningfully less than poor blood glucose control by 57.4% with a mean difference of 102.56 mg/dl between the groups. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real reduction in post prandial blood glucose levels among good blood glucose control group

Duration of Diabetes

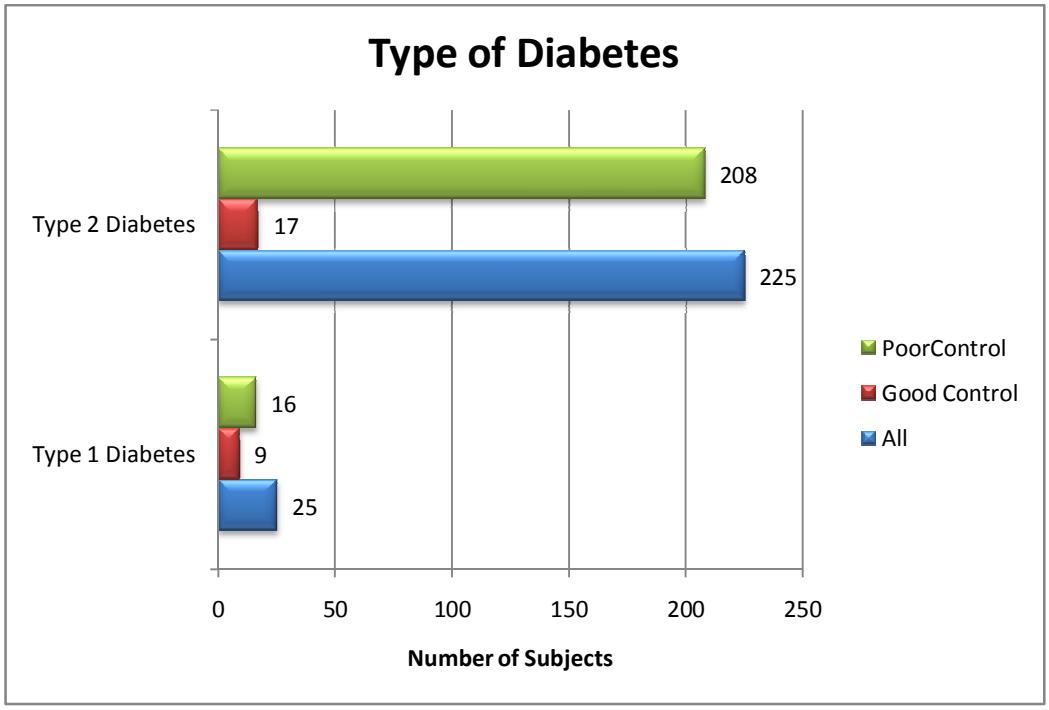


Duration of Diabetes	All	%	Good Control	%	Poor Control	%
≤ 2 years	41	16.4	4	15.38462	38	16.96429
2 to 5 years	60	24	8	30.76923	52	23.21429
5 to 10 years	99	39.6	10	38.46154	89	39.73214
> 10 years	49	19.6	4	15.38462	45	20.08929
Total	249	99.6	26	100	224	100

Duration of Diabetes	All	Good Control	Poor Control
N	249	26	224
Mean	7.343373	6.403846	7.419643
SD	4.688649	3.77894	4.793752
P value Unpaired t test		0.2166	

By conventional criteria the association between the study groups and duration of diabetes is considered to be not statistically significant since $p > 0.05$.

Type of Diabetes



Type of Diabetes	All	%	Good Control	%	PoorControl	%
Type 1 Diabetes	25	10	9	34.61538	16	7.142857
Type 2 Diabetes	225	90	17	65.38462	208	92.85714
Total	250	100	26	100	224	100
P value Chi squared Test	0.00001					

By conventional criteria the association between the study groups and type of diabetes among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the mucocutaneous lesions among diabetic patients, type 2 diabetes patients in good blood glucose control group is predominantly lesswhen compared to poor blood glucose control group. It is statistically significant with a p-value of 0.00001 according to chi squared test.

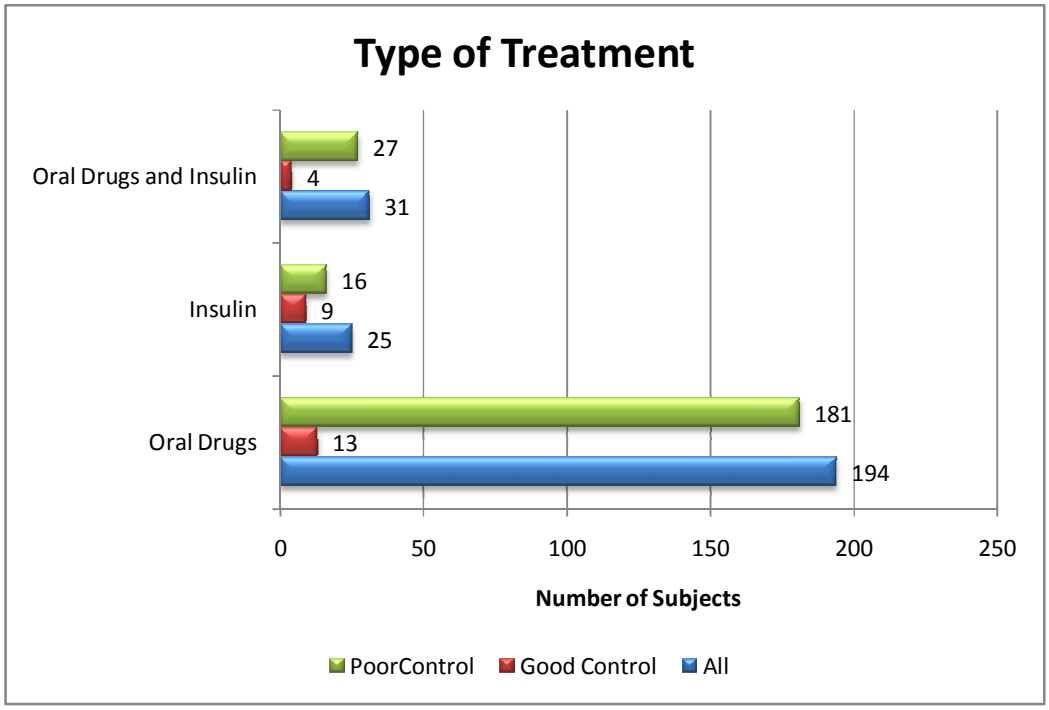
Clinical Significance

The , the percentage of type 2 diabetes patients in good blood glucose control group is meaningfully less in good blood glucose control group than poor blood glucose control by 42% with a difference of 27.47 percentage points between the groups. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in , type 2 diabetes patients among poor blood glucose control group

Type of Treatment



Type of Treatment	All	%	Good Control	%	PoorControl	%
Oral Drugs	194	77.6	13	50	181	80.80357
Insulin	25	10	9	34.61538	16	7.142857
Oral Drugs and Insulin	31	12.4	4	15.38462	27	12.05357
Total	250	100	26	100	224	100
P value Chi squared Test	0.00000					

By conventional criteria the association between the study groups and type of treatment among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the mucocutaneous lesions among diabetic patients, the treatment with oral drugs given to patients in good blood glucose control group is predominantly less when compared to poor blood glucose control group. Similarly the treatment with insulin given to patients in good blood glucose control group is predominantly high when compared to poor blood glucose control group It is statistically significant with a p-value of 0.00000 according to chi squared test.

Clinical Significance

The percentage of patients on oral drugsin good blood glucose control group is meaningfully less in good blood glucose control group than poor blood glucose control by 1.61 times with a difference of 30.80 percentage points between the groups.

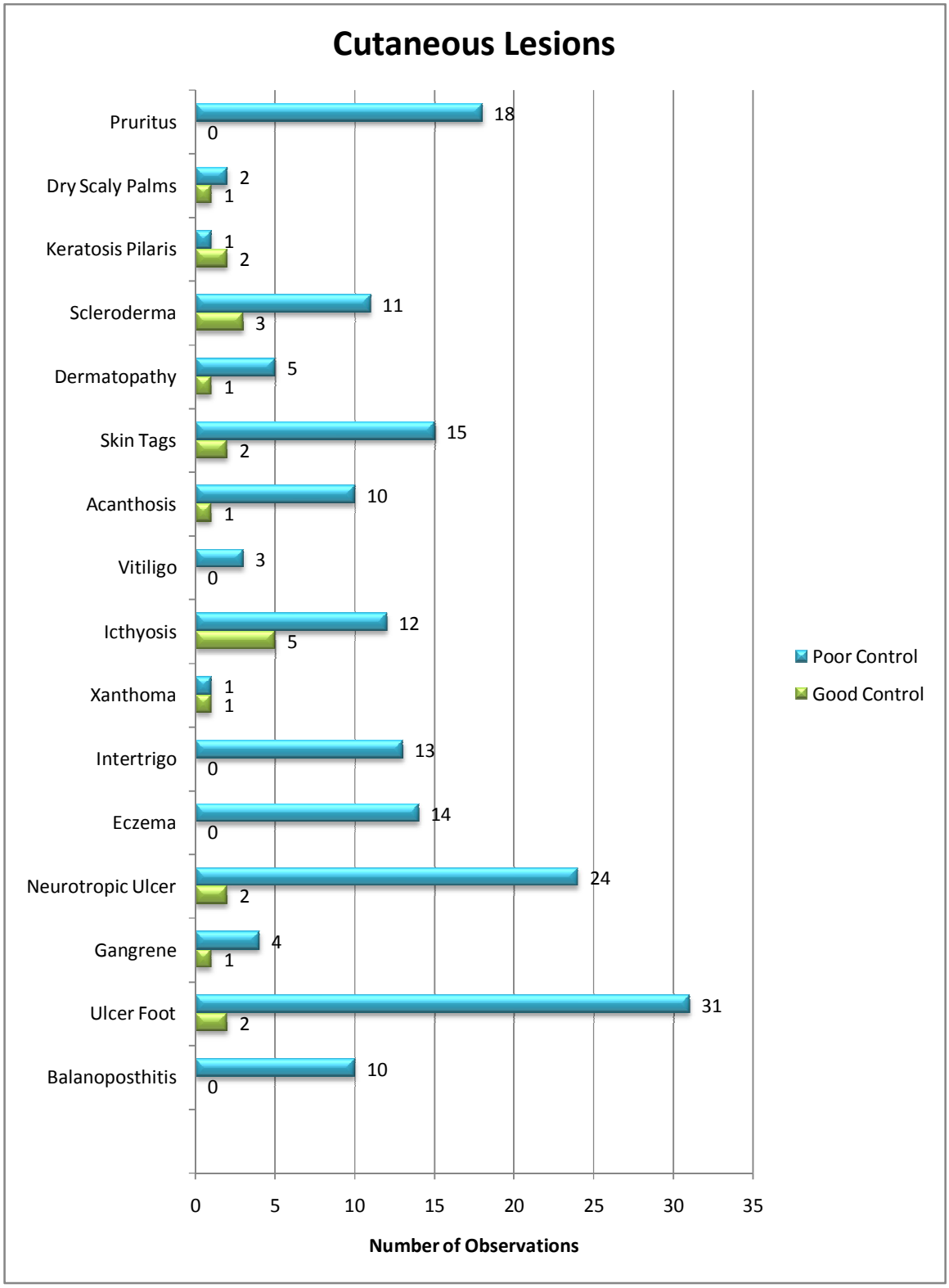
The percentage of patients on insulin in good blood glucose control group is meaningfully more than poor blood glucose control by 4.85 times with a difference of 27.47 percentage points between the groups.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in patients with good blood glucose control when on insulin and meaningfully real increase in patients with poor blood glucose control when on oral drugs.

Cutaneous Lesions



Cutaneous Lesions	All	%	Good Control	%	Poor Control	%	P value Chi squared Test
Balanoposthitis	10	4	0	0	10	4.464286	0.271515
Ulcer Foot	33	13.2	2	7.692308	31	13.83929	0.380754
Gangrene	5	2	1	3.846154	4	1.785714	0.477487
Neuropathic Ulcer	26	10.4	2	7.692308	24	10.71429	0.632781
Eczema	14	5.6	0	0	14	6.25	0.189513
Intertrigo	13	5.2	0	0	13	5.803571	0.207084
Xanthoma	2	0.8	1	3.846154	1	0.446429	0.065479
Icthyosis	17	6.8	5	19.23077	12	5.357143	0.007816
Vitiligo	3	1.2	0	0	3	1.339286	0.552733
Acanthosis	11	4.4	1	3.846154	10	4.464286	0.884342
Skin Tags	17	6.8	2	7.692308	15	6.696429	0.848577
Dermapathy	6	2.4	1	3.846154	5	2.232143	0.610753
Scleroderma	14	5.6	3	11.53846	11	4.910714	0.164129
Keratosis Pilaris	3	1.2	2	7.692308	1	0.446429	0.001319
Dry Scaly Palms	3	1.2	1	3.846154	2	0.892857	0.190495
Pruritus	18	7.2	0	0	18	8.035714	0.133495

Cutaneous Lesions	All	Good Control	PoorControl
Absent	72	3	69
Present	178	26	152
Total	250	29	221
P value Chi squared Test	0.019581		

By conventional criteria the association between the study groups and cutaneous lesions among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the mucocutaneous lesions among diabetic patients, the prevalence of cutaneous lesions among patients in good blood glucose control group is predominantly more when compared to poor blood glucose control group. It is statistically significant with a p-value of 0.019581 according to chi squared test.

Clinical Significance

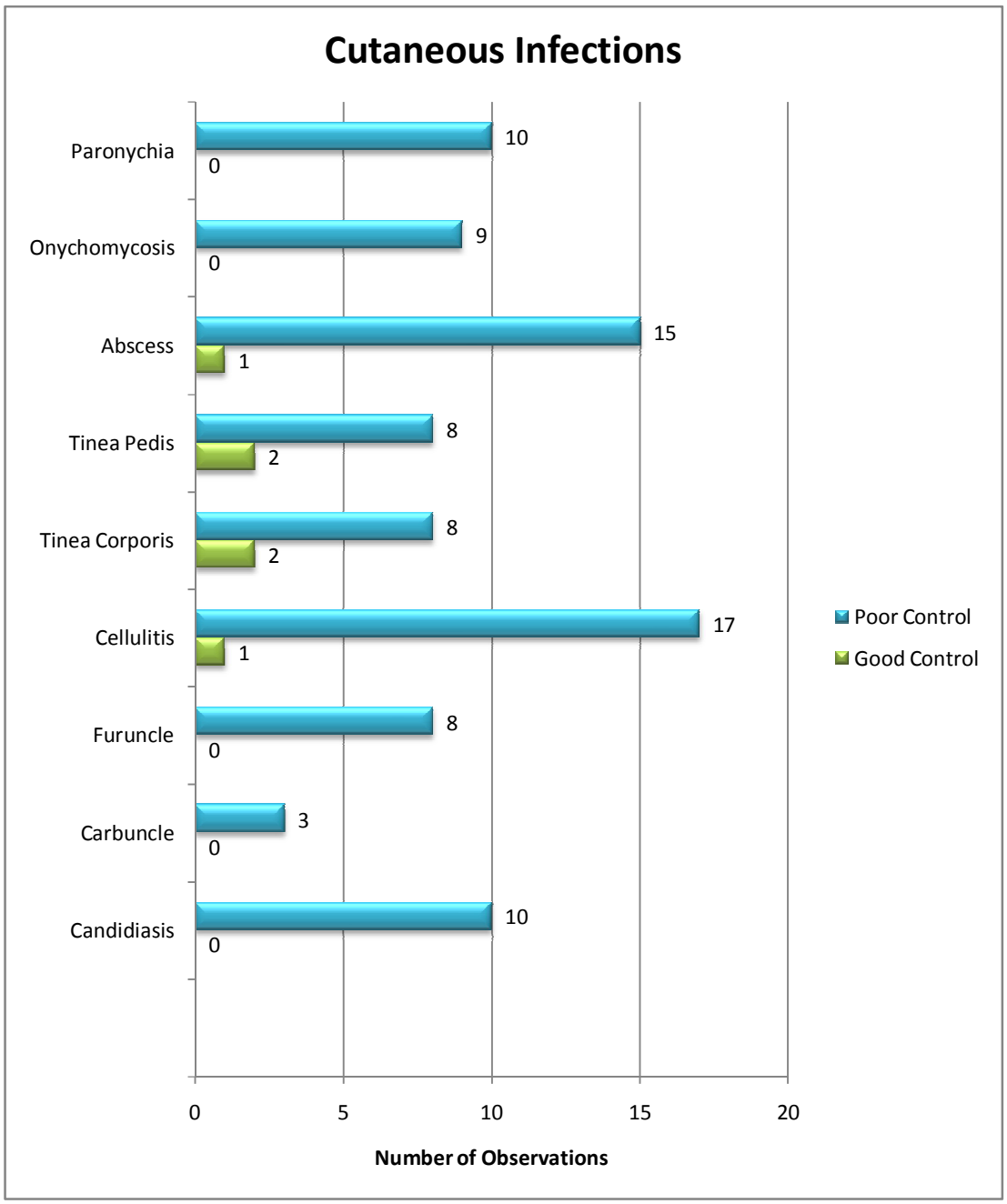
The prevalence of cutaneous lesions among patientsin good blood glucose control group is meaningfully more in good blood glucose control group than poor blood glucose control by 1.3 times with a difference of 20.88 percentage points between the groups.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in patients with cutaneous lesions among good blood glucose control when compared with poor blood glucose control.

Cutaneous Infections



Cutaneous Infections	All	%	Good Control	%	Poor Control	%	P value Chi squared Test
Candidiasis	13	5.2	0	0	10	4.464286	0.268165
Carbuncle	3	1.2	0	0	3	1.339286	0.552733
Furuncle	8	3.2	0	0	8	3.571429	0.327371
Cellulitis	18	7.2	1	3.846154	17	7.589286	0.484594
TineaCorporis	10	4	2	7.692308	8	3.571429	0.310107
TineaPedis	10	4	2	7.692308	8	3.571429	0.310107
Abscess	16	6.4	1	3.846154	15	6.696429	0.57406
Onychomycosis	9	3.6	0	0	9	4.017857	0.297881
Paronychia	10	4	0	0	10	4.464286	0.271515

Cutaneous Infections	All	Good Control	PoorControl
Absent	155	26	129
Present	95	3	92
Total	250	29	221
P value Chi squared Test	0.001101		

By conventional criteria the association between the study groups and cutaneous infections among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the mucocutaneous lesions among diabetic patients, the prevalence of cutaneous infections among patients in good blood glucose control group is predominantly less when compared to poor blood glucose control group. It is statistically significant with a p-value of 0.001101 according to chi squared test.

Clinical Significance

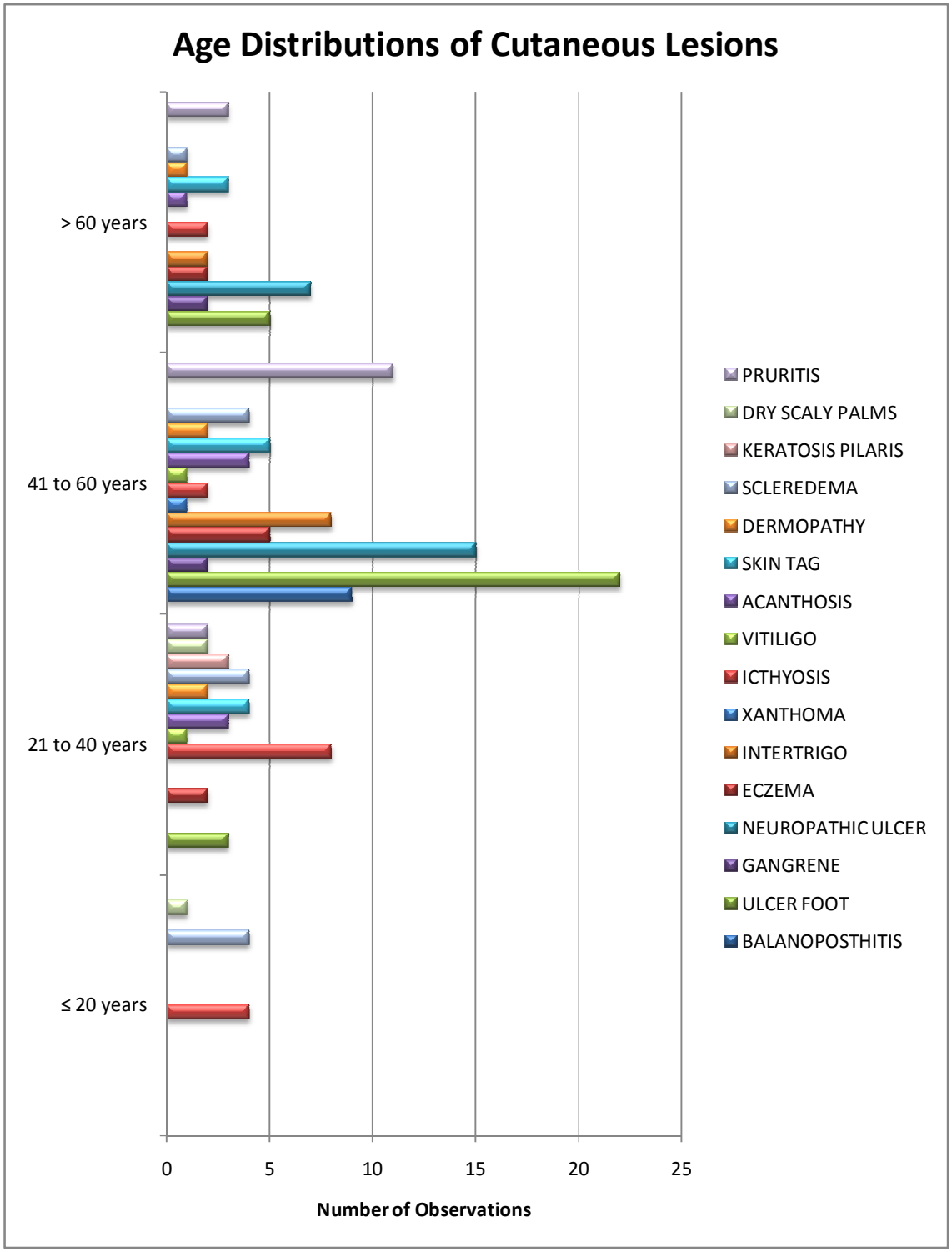
The prevalence of cutaneous infections among patients in good blood glucose control group is meaningfully less in good blood glucose control group than poor blood glucose control by 14.02 times with a difference of 31.28 percentage points between the groups.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real decrease in patients with cutaneous infections among good blood glucose control when compared with poor blood glucose control.

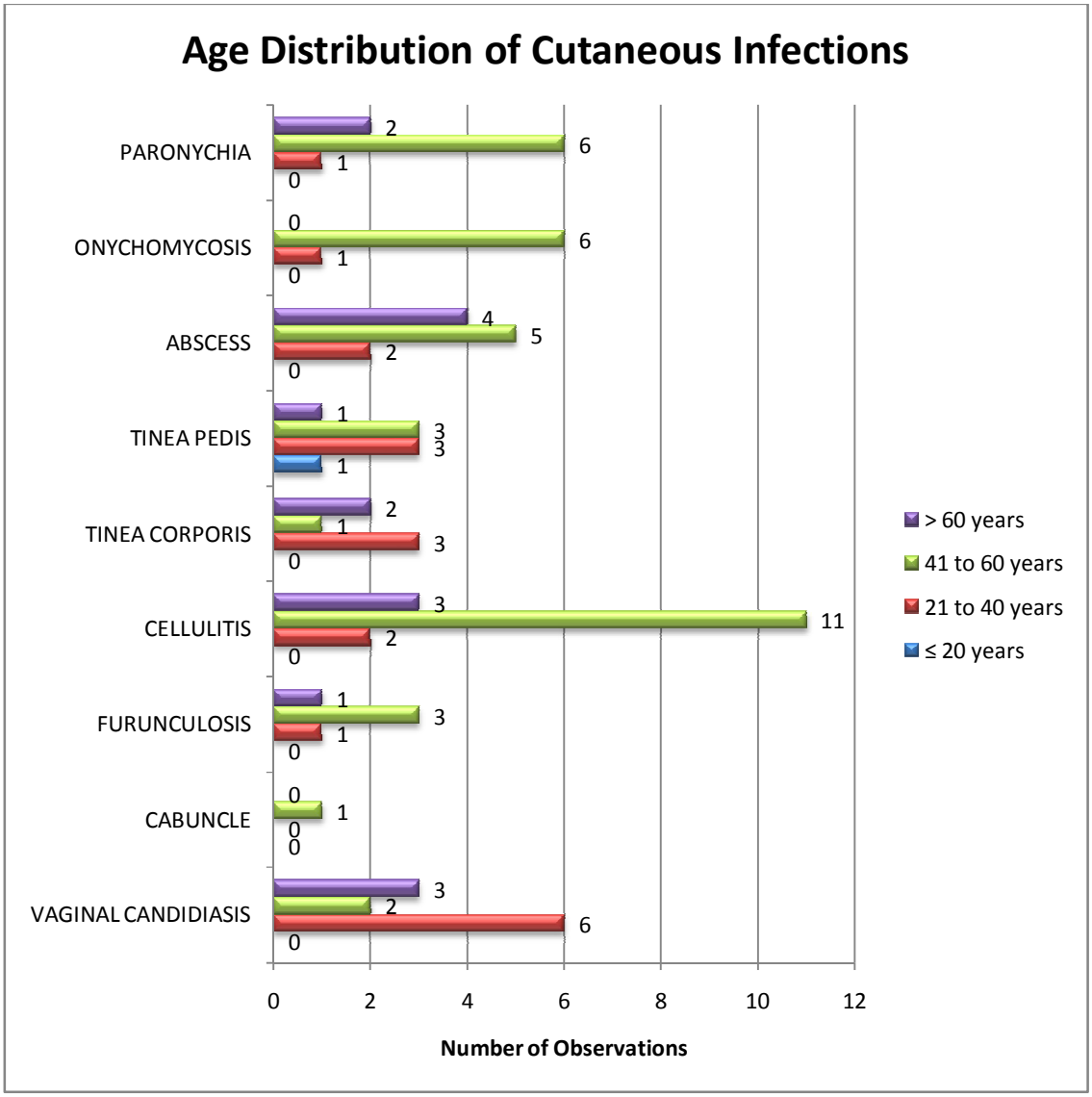
Age Distributions of Cutaneous Lesions



Age Distribution of Cutaneous Lesions	≤ 20 years	21 to 40 years	41 to 60 years	> 60 years	Total	%
BALANOPOSTHITIS	0	0	9	0	9	6
ULCER FOOT	0	3	22	5	30	18
GANGRENE	0	0	2	2	4	2
NEUROPATHIC ULCER	0	0	15	7	22	13
ECZEMA	0	2	5	2	9	6
INTERTRIGO	0	0	8	2	10	6
XANTHOMA	0	0	1	0	1	1
ICTHYOSIS	4	8	2	2	16	10
VITILIGO	0	1	1	0	2	1
ACANTHOSIS	0	3	4	1	8	5
SKIN TAG	0	4	5	3	12	7
DERMOPATHY	0	2	2	1	5	3
SCLEREDEMA	4	4	4	1	13	8
KERATOSIS PILARIS	0	3	0	0	3	2
DRY SCALY PALMS	1	2	0	0	3	2
PRURITUS	0	2	11	3	16	10
Total					163	100
P value Chi squared Test					0.9820	

By conventional criteria the association between the age groups and distribution of cutaneous lesions is considered to be not statistically significant since $p > 0.05$.

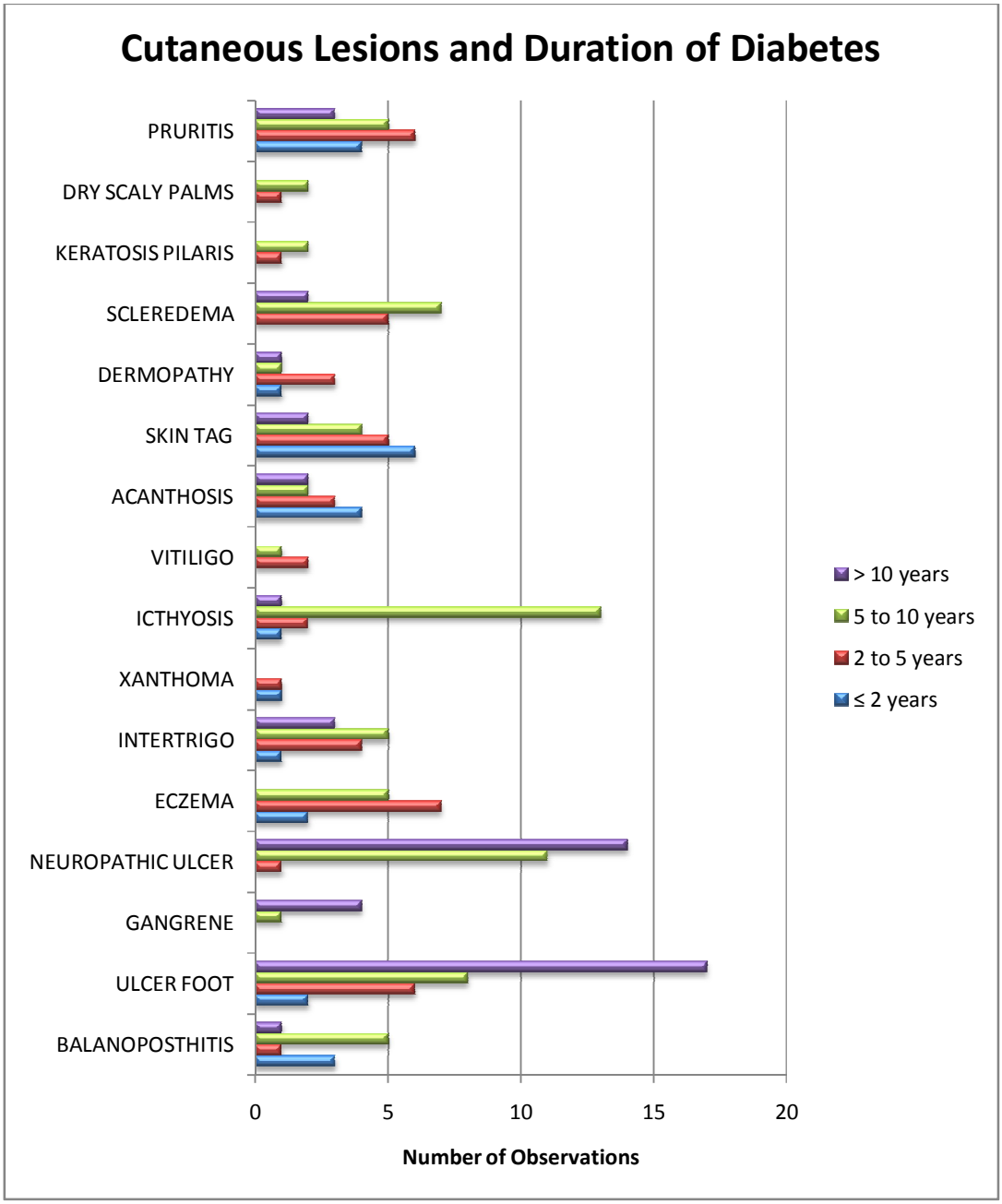
Age Distribution of Cutaneous Infections



Age Distribution of Cutaneous Infections	≤ 20 years	21 to 40 years	41 to 60 years	> 60 years	Total	%
VAGINAL CANDIDIASIS	0	6	2	3	11	15
CARBUNCLE	0	0	1	0	1	1
FURUNCULOSIS	0	1	3	1	5	7
CELLULITIS	0	2	11	3	16	22
TINEA CORPORIS	0	3	1	2	6	8
TINEA PEDIS	1	3	3	1	8	11
ABSCESS	0	2	5	4	11	15
ONYCHOMYCOSIS	0	1	6	0	7	9
PARONYCHIA	0	1	6	2	9	12
Total					74	100
P value Chi squared Test					0.4769	

By conventional criteria the association between the age groups and distribution of cutaneous infections is considered to be not statistically significant since $p > 0.05$.

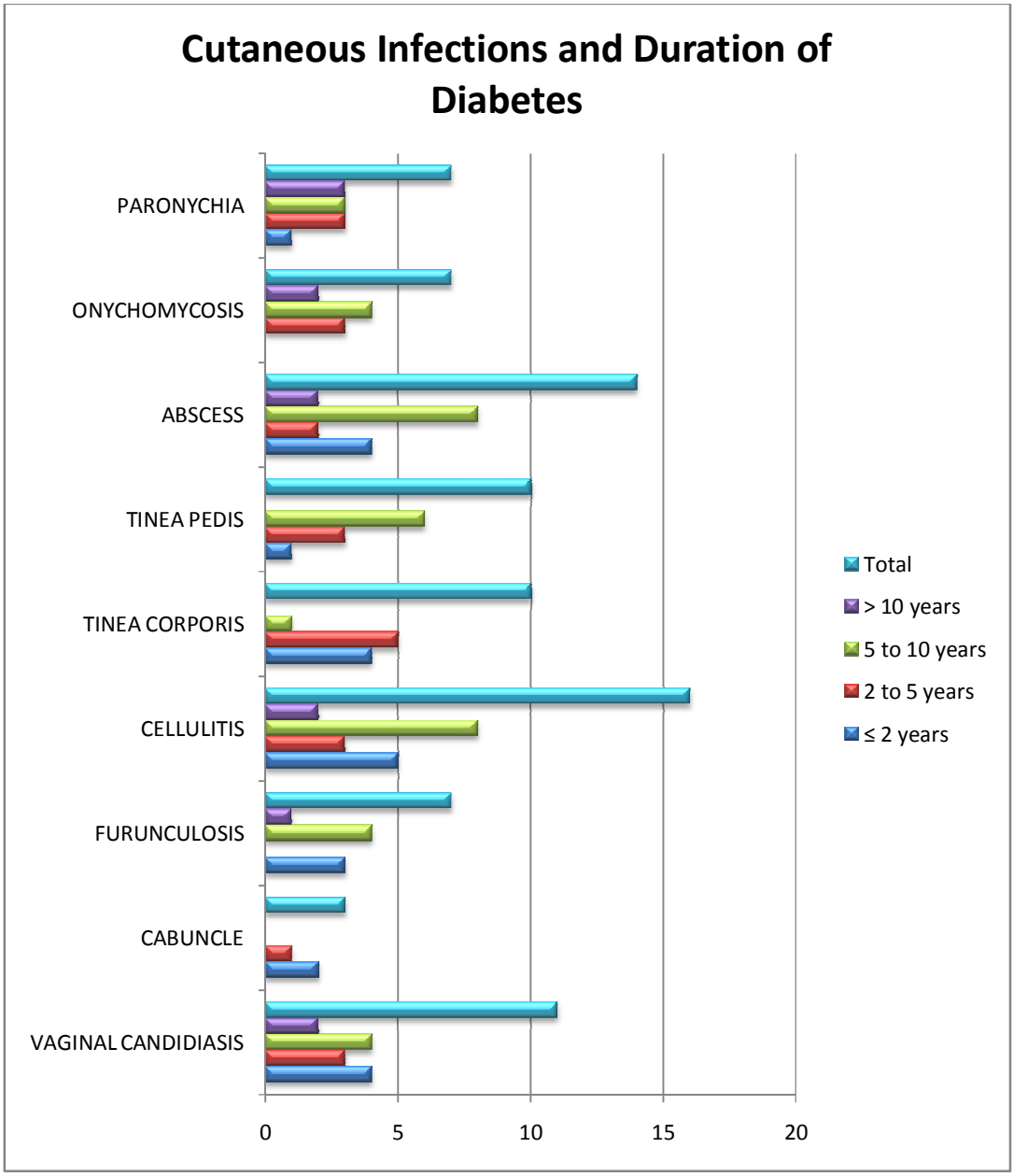
Cutaneous Lesions and Duration of Diabetes



Duration of Diabetes and Cutaneous Lesions	≤ 2 years	2 to 5 years	5 to 10 years	> 10 years	Total	%
BALANOPOSTHITIS	3	1	5	1	9	6
ULCER FOOT	2	6	8	17	16	11
GANGRENE	0	0	1	4	1	1
NEUROPATHIC ULCER	0	1	11	14	12	8
ECZEMA	2	7	5	0	14	10
INTERTRIGO	1	4	5	3	10	7
XANTHOMA	1	1	0	0	2	1
ICTHYOSIS	1	2	13	1	16	11
VITILIGO	0	2	1	0	3	2
ACANTHOSIS	4	3	2	2	9	6
SKIN TAG	6	5	4	2	15	10
DERMOPATHY	1	3	1	1	5	3
SCLEREDEMA	0	5	7	2	12	8
KERATOSIS PILARIS	0	1	2	0	3	2
DRY SCALY PALMS	0	1	2	0	3	2
PRURITUS	4	6	5	3	15	10
Tamil					145	100
P value Chi squared Test					0.7766	

By conventional criteria the association between the duration of diabetes and distribution of cutaneous lesions is considered to be not statistically significant since $p > 0.05$.

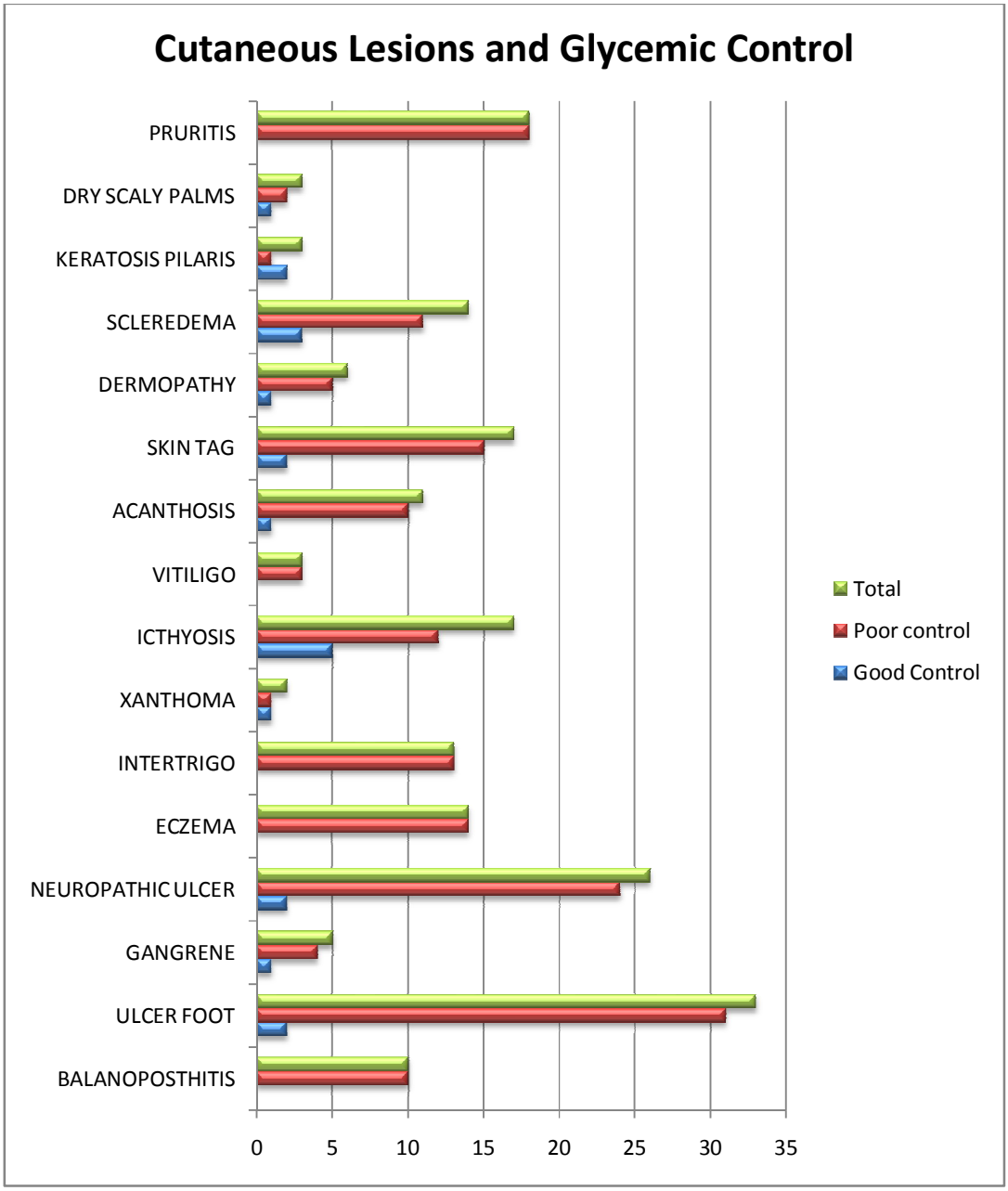
Cutaneous Infections and Duration of Diabetes



Duration of Diabetes and Cutaneous Infections	≤ 2 years	2 to 5 years	5 to 10 years	> 10 years	Total	%
VAGINAL CANDIDIASIS	4	3	4	2	11	13
CARBUNCLE	2	1	0	0	3	4
FURUNCULOSIS	3	0	4	1	7	8
CELLULITIS	5	3	8	2	16	19
TINEA CORPORIS	4	5	1	0	10	12
TINEA PEDIS	1	3	6	0	10	12
ABSCESS	4	2	8	2	14	16
ONYCHOMYCOSIS	0	3	4	2	7	8
PARONYCHIA	1	3	3	3	7	8
Total					85	100
P value Chi squared Test					0.0924	

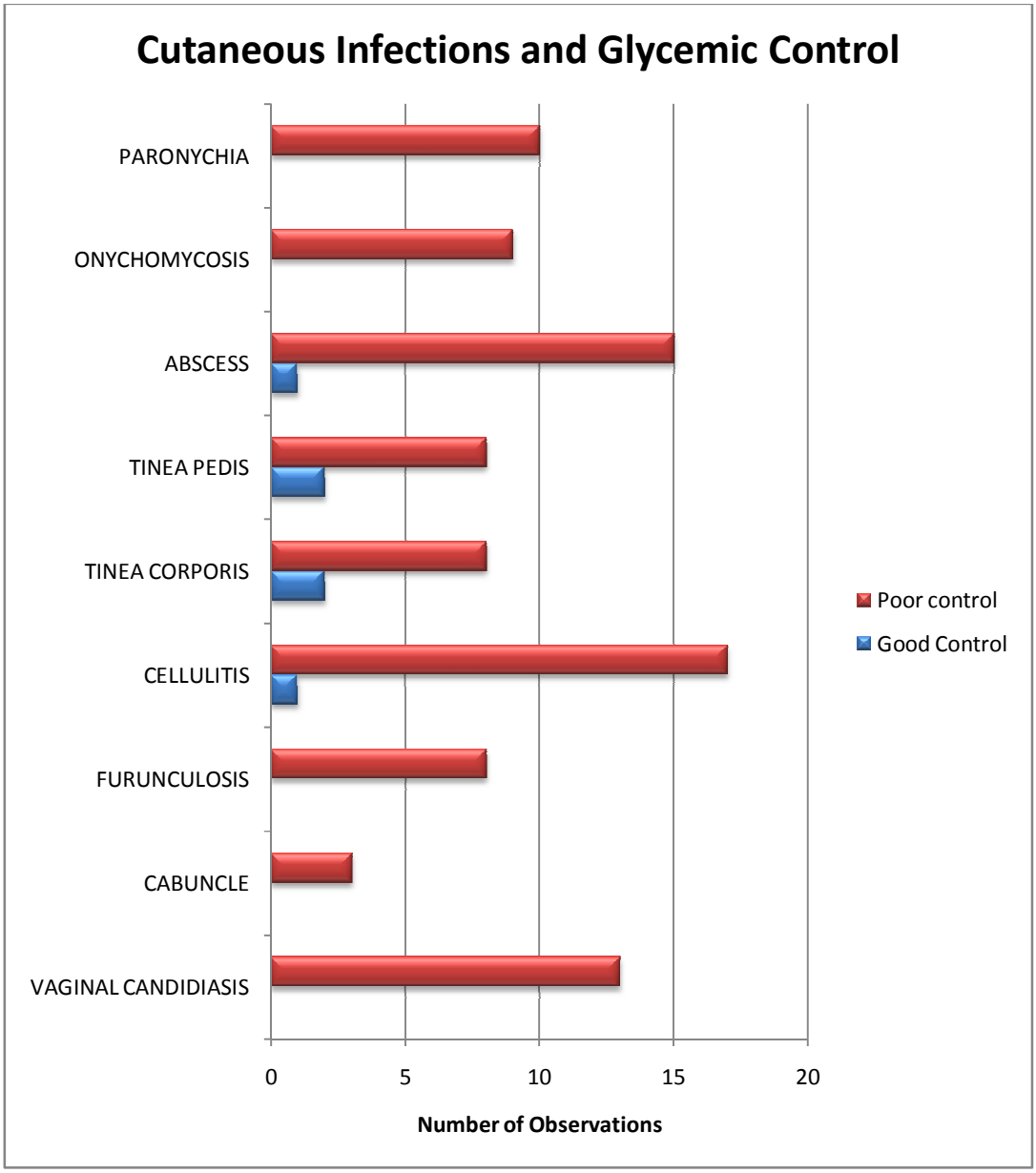
By conventional criteria the association between the duration of diabetes and distribution of cutaneous infections is considered to be not statistically significant since $p > 0.05$.

Cutaneous Lesions and Glycemic Control

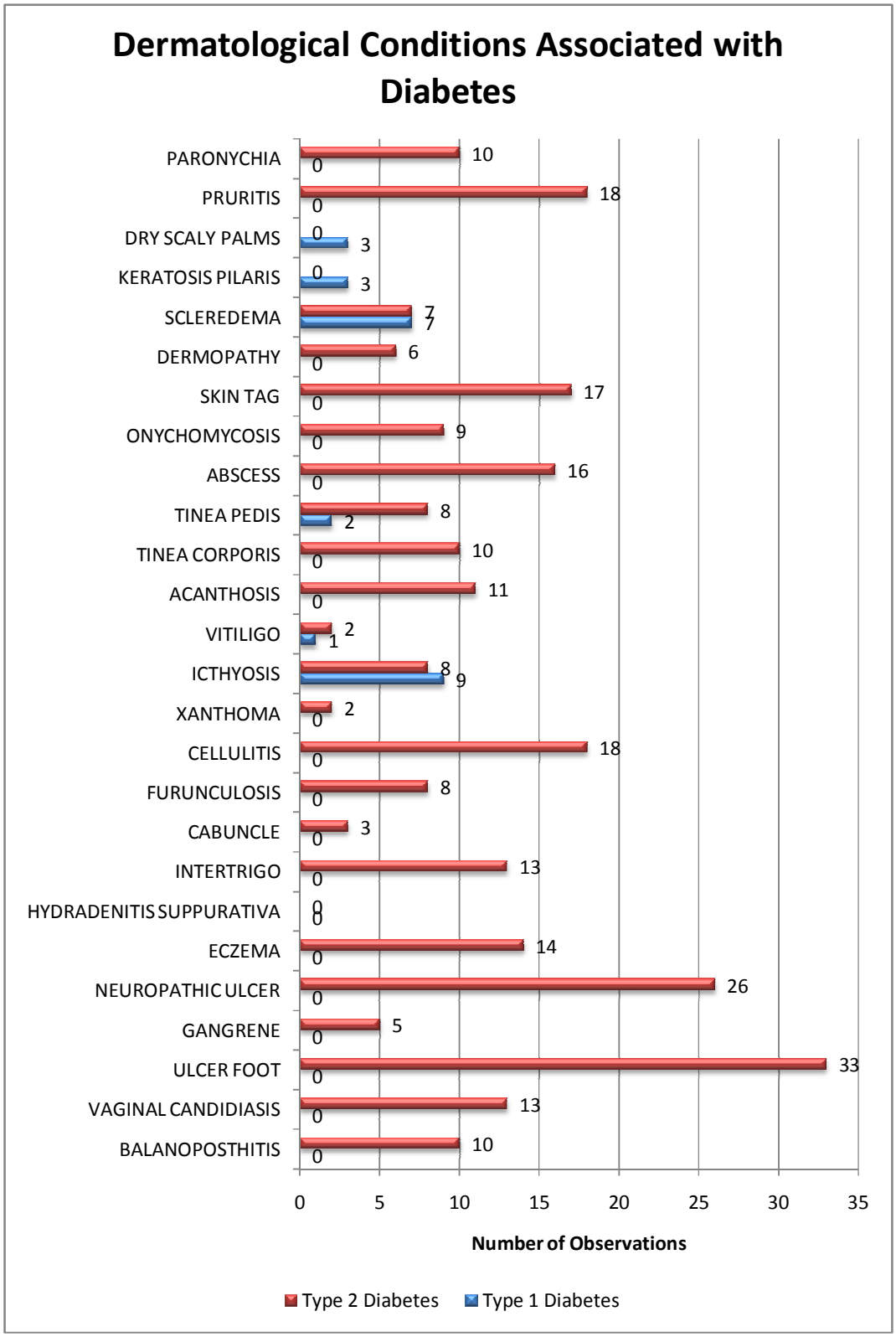


Glycemic Control and Cutaneous Lesions	Good Control	Poor control	Total	%
BALANOPOSTHITIS	0	10	10	5
ULCER FOOT	2	31	33	17
GANGRENE	1	4	5	3
NEUROPATHIC ULCER	2	24	26	13
ECZEMA	0	14	14	7
INTERTRIGO	0	13	13	7
XANTHOMA	1	1	2	1
ICTHYOSIS	5	12	17	9
VITILIGO	0	3	3	2
ACANTHOSIS	1	10	11	6
SKIN TAG	2	15	17	9
DERMOPATHY	1	5	6	3
SCLEREDEMA	3	11	14	7
KERATOSIS PILARIS	2	1	3	2
DRY SCALY PALMS	1	2	3	2
PRURITUS	0	18	18	9
Total			195	100

Cutaneous Infections and Glycemic Control



Glycemic Control and Cutaneous Infections	Good Control	Poor control	Total	%
VAGINAL CANDIDIASIS	0	13	13	13
CARBUNCLE	0	3	3	3
FURUNCULOSIS	0	8	8	8
CELLULITIS	1	17	18	19
TINEA CORPORIS	2	8	10	10
TINEA PEDIS	2	8	10	10
ABSCESS	1	15	16	16
ONYCHOMYCOSIS	0	9	9	9
PARONYCHIA	0	10	10	10
Total			97	100


Dermatological Conditions Associated with Diabetes
PARONYCHIA
PRURITIS
DRY SCALY PALMS
KERATOSIS PILARIS
SCLEREDEMA
DERMOPATHY
SKIN TAG
ONYCHOMYCOSIS
ABSCESS
TINEA PEDIS
TINEA CORPORIS
ACANTHOSIS
VITILIGO
ICTHYOSIS
XANTHOMA
CELLULITIS
FURUNCULOSIS
CABUNCLE
INTERTRIGO
HYDRADENITIS SUPPURATIVA
ECZEMA
NEUROPATHIC ULCER
GANGRENE
ULCER FOOT
VAGINAL CANDIDIASIS
BALANOPOSTHITIS
10
0
18
0
0
3
0
3
7
7
6
0
17
0
9
0
16
0
8
2
10
0
11
0
2
1
8
9
2
0
18
0
8
0
3
0
13
0
0
0
14
0
26
0
5
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33
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10
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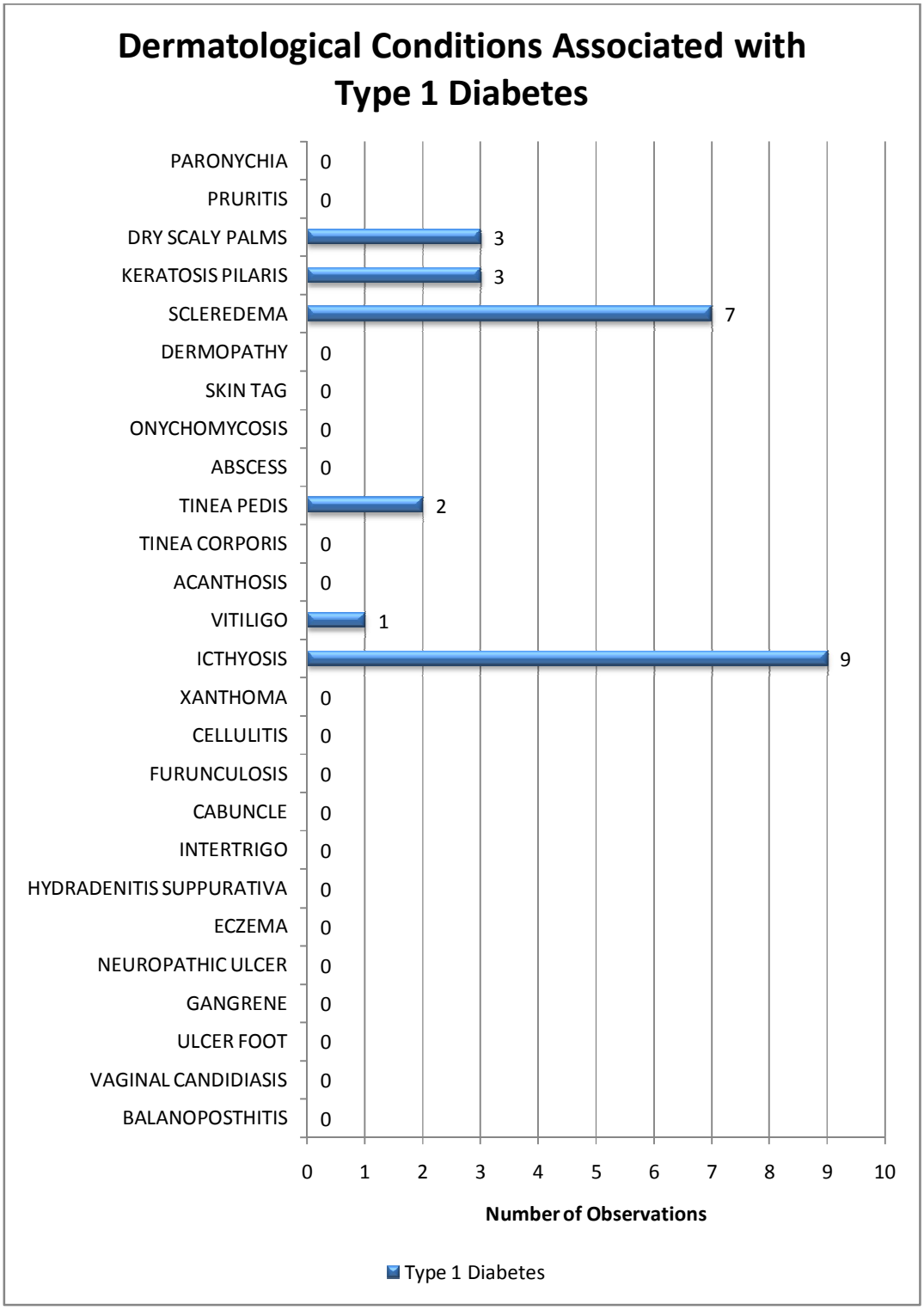
0
5
10
15
20
25
30
35
Number of Observations
Type 2 Diabetes
Type 1 Diabetes


Dermatological Conditions Associated with Type 1 Diabetes
PARONYCHIA
PRURITIS
DRY SCALY PALMS
KERATOSIS PILARIS
SCLEREDEMA
DERMOPATHY
SKIN TAG
ONYCHOMYCOSIS
ABSCESS
TINEA PEDIS
TINEA CORPORIS
ACANTHOSIS
VITILIGO
ICTHYOSIS
XANTHOMA
CELLULITIS
FURUNCULOSIS
CABUNCLE
INTERTRIGO
HYDRADENITIS SUPPURATIVA
ECZEMA
NEUROPATHIC ULCER
GANGRENE
ULCER FOOT
VAGINAL CANDIDIASIS
BALANOPOSTHITIS
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3
3
7
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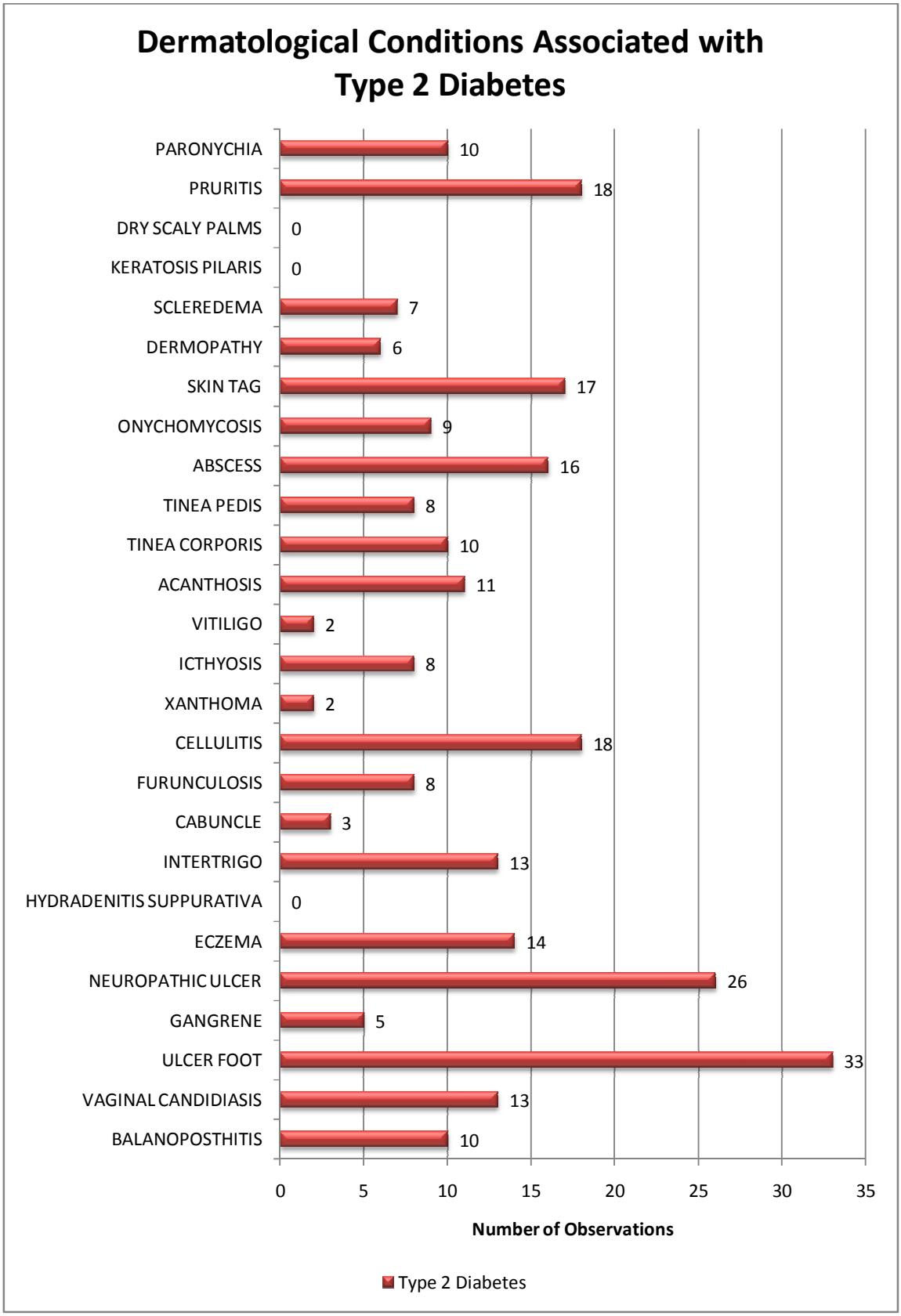
0
2
0
0
1
9
0
0
0
0
0
0
0
0
0
0
0
0
0 1 2 3 4 5 6 7 8 9 10
Number of Observations
Type 1 Diabetes


Dermatological Conditions Associated with Type 2 Diabetes
PARONYCHIA
PRURITIS
DRY SCALY PALMS
KERATOSIS PILARIS
SCLEREDEMA
DERMOPATHY
SKIN TAG
ONYCHOMYCOSIS
ABSCESS
TINEA PEDIS
TINEA CORPORIS
ACANTHOSIS
VITILIGO
ICTHYOSIS
XANTHOMA
CELLULITIS
FURUNCULOSIS
CABUNCLE
INTERTRIGO
HYDRADENITIS SUPPURATIVA
ECZEMA
NEUROPATHIC ULCER
GANGRENE
ULCER FOOT
VAGINAL CANDIDIASIS
BALANOPOSTHITIS
10
18
0
0
7
6
17
9
16
8
10
11
2
8
2
18
8
3
13
0
14
26
5
33
13
10
0
5
10
15
20
25
30
35
Number of Observations
Type 2 Diabetes

Dermatological Conditions	Type 1 Diabetes	Type 2 Diabetes	Total	%
BALANOPOSTHITIS	0	10	10	3.42
VAGINAL CANDIDIASIS	0	13	13	4.45
ULCER FOOT	0	33	33	11.30
GANGRENE	0	5	5	1.71
NEUROPATHIC ULCER	0	26	26	8.90
ECZEMA	0	14	14	4.79
HYDRADENITIS SUPPURATIVA	0	0	0	0.00
INTERTRIGO	0	13	13	4.45
CABUNCLE	0	3	3	1.03
FURUNCULOSIS	0	8	8	2.74
CELLULITIS	0	18	18	6.16
XANTHOMA	0	2	2	0.68
ICTHYOSIS	9	8	17	5.82
VITILIGO	1	2	3	1.03
ACANTHOSIS	0	11	11	3.77
TINEA CORPORIS	0	10	10	3.42
TINEA PEDIS	2	8	10	3.42
ABSCESS	0	16	16	5.48
ONYCHOMYCOSIS	0	9	9	3.08
SKIN TAG	0	17	17	5.82
DERMOPATHY	0	6	6	2.05
SCLEREDEMA	7	7	14	4.79
KERATOSIS PILARIS	3	0	3	1.03
DRY SCALY PALMS	3	0	3	1.03
PRURITUS	0	18	18	6.16
PARONYCHIA	0	10	10	3.42
Total	25	267	292	100

DISCUSSION

Previous studies shows that example

Kahana Et al screened 216 patients with skin tags and found to have overt Diabetes mellitus and impaired glucose tolerance test is 7.9%

Thappa Et al found that 62% of patients with skin tags has diabetes. So these 2 studies state that these are markers for diabetes mellitus.

Requena Et al found that clear cell porocarcinoma as a cutaneous marker of diabetes mellitus. Glycogen accumulation with in their cytoplasm results in clear cell appearance of neoplastic cells.

Grandhe Et al found that AN is independent cutaneous marker of T2DM. There is also increased frequency of diabetic dermopathy, hairloss over legs, AN, syringoma, callosity, brittle nails, icthyosis, cutaneous amyloidosis.

Mahajen Et al shows no statistical association between diabetic dermoangiopathy and diabetic retinopathy, neuropathy, hypertension.

AN is present in 3% of the diabetics.Pruritus is present in 10 % of diabetics. Most common age group is 41-50 years.

Frost Et al shows 33% had LJM in 335 type 1 diabetes mellitus.

In mutairi Et al most common age group is 40-60 years. AN is present in 4.7%

Nigam and pande Et al shows 61% of diabetics had cutaneous dermatosis and 4.5% had pruritus.

In bhat Et al duration of diabetes is upto 10 years. AN is present in 5.3% of Diabetics.

In Ahmed Et al, AN is present in 2.8% of Diabetics.

Shivanna Ragunatha Et al shows majority of patients have FBG < 130% and type 2 DM is 60%. Statistically significant difference between the patients with and without DM specific cutaneous disorders was noticed with reference to age and gender distribution, duration of DM and FBG.

Signs of IR, acrochordan 26% , AN 5% , Fungal infection 13%, bacterial infection 6.8%, eruptive xanthoma 0.6%, diabetic foot 0.2%, diabetic bulla 0.4%, diabetic dermopathy 0.2%, generalized granuloma annulare 0.2%, insulin reaction 6.2%, lipo dystrophy 14%.

DuriyeDenizdermiseren Et al shows 750 patients have examined of these most common cutaneous skin manifestations are cutaneous infections 47%, xerosis 26%, inflammatory skin disease 20%. Patients with HbA1c greater than or equal to 8 mmol had more skin disorders than HbA1C of less than 8 mmol.

SUMMARY AND CONCLUSION

Diabetic Patients who are attending Diabetology OPD, Dermatology OPD & ward, Medicine OPD & ward were screened for dermatological manifestations. Patients height, weight, BMI, Blood pressure was measured. History of Alcohol & Smoking was obtained. Blood investigations (FBS, PPBS, RFT) were done. History regarding Duration of diabetes and associated medical illness were also obtained. Treatment details regarding DM were also recorded. Dermatologist opinion was obtained. Patient's muco cutaneous manifestations were treated according to guidelines.

In this study, in Type 2 DM , most of muco cutaneous manifestations(Eg. Cellulitis, vaginal candidiasis, abscess, paronychia ,scleredema, eczema, intertrigo) are associated with poor glycemic status.

Cutaneous infections are associated with poor glucose control.

Neuropathic ulcer, ulcer foot, Abscess and cellulitis are associated with long duration of diabetes (>10years duration).

In newly detected DM, common manifestations are furunculosis, pruritus, abscess,skin tag, tinea corporis, acanthosis, vaginal candidiasis, balanoposthitis.

Most common age group for mucocutaneous manifestations are 41-60years. Most of the mucocutaneous manifestations commonly occur in 5-10 yrs of duration of diabetes.

In Type1DM, distribution of mucocutaneous manifestations are

Icthyosis	36%
Keratosis pilaris	12%
Dry scaly palms	12%
Vitiligo	4%
Sceleroderma like changes	28%
Tinea pedis	8%

In Type 2 DM most common mucocutaneous manifestations are ulcer foot (11.3%)

Demographic profile	number	percentage
Total patients	250	100
Type 1- male	10	4
Type 1- female	15	6
Type 2 - male	100	40
Type 2 – female	125	50

Type 1 DM (Uncontrolled)	22	8.8
Type 1 DM (controlled)	3	1.2
Type 2 DM (Uncontrolled)	202	80.8
Type 2 DM (Controlled)	23	9.2
Mean age - type 1	24+/-4	
Mean age - type 2	52+/-7	
Mean Duration in years - type 1	7 +/- 1	
Mean Duration in years - type 2	8+/- 5	

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PROFORMA

Name:

Age/sex:

Occupation: DOB:

Address with contact no:

IP NO/OP NO:

Type of diabetes:

Treatment history:

History of Mucocutaneous manifestations:

Family history of skin manifestations:

FBS: PPBS: HIV:

B.Urea S.Creatinine

Height: Weight: BMI:

PAST HISTORY

Hypertension: CAD: Epilepsy:

Dyslipidemia: CKD: CVA:

EXAMINATION:

Pallor: Icterus:

Pedal edema:

PR: BP:

CVS:

RS:

P/A:

CNS:

MUCOCUTANEOUS MANIFESTATIONS

CUTANEOUS INFECTIONS

Candidiasis

Intertrigo

Folliculitis

Furunculosis

Carbuncle

Cellulitis

Malignant otitis externa

Paronychia

Onychomycosis

Abscess

Eczema

Tinea pedis

Tinea Corporis

Gangrene

Ulcer foot

Vaginal Candidiasis

Balanoposthitis

NEUROLOGIC LESIONS

Neuropathic ulcers

DISORDERS OF COLLAGEN

Necrobiosis lipoidica

Granuloma annulare

Scleredema diabeticorum

Scleroderma like change of the hand

Waxy skin

METABOLIC DISEASE

Xanthomatosis

Generalized pruritus

OTHERS

Acquired icthyosis Diabetic dermopathy

Diabetic bullae Vitiligo

Acanthosis nigricans Xanthoma Skin Tag

MASTER CHART

Serial No.	AGE	SEX	Ht in mt	Wt in kgs	BMI	alcohol	smoking	BP	FBS	PPBS	duration of diabetes	skin findings	type of DM	treatment
1	60	F	1.7	65	22.4	NO	NO	120/80	170	278	5	ulcer foot	t2	o
2	57	f	1.6	85	32.0	NO	NO	150/90	320	450	10	cellulitis, pruritus	t2	o+i
3	43	f	1.40	55	28.1	NO	NO	120/70	98	196	1	skin tags	t2	o
4	60	f	1.55	60	25.0	NO	NO	130/90	126	224	2	acanthosis	t2	o
5	48	f	1.7	65	27.1	NO	NO	130/80	152	252	5	ulcer foot,paronychia	t2	o
6	44	f	1.7	55	26.2	NO	NO	140/90	178	300	5	vitiligo	t2	o
7	57	f	1.45	55	26.2	NO	NO	150/80	112	198	5	acanthosis	t2	o
8	50	f	1.55	60	25.0	NO	NO	150/90	106	186	5	skin tag	t2	o
9	53	f	1.45	55	26.2	NO	NO	130/90	145	300	3	ulcer foot	t2	o
10	65	f	1.53	55	23.5	NO	NO	120/80	156	256	15	dermopathy	t2	o+i
11	65	f	1.45	70	33.3	NO	NO	140/90	102	196	15	icthyosis	t2	o+i
12	56	f	1.6	63	24.6	NO	NO	130/80	238	357	newly detected	cellulitis,pruritus	t2	o
13	47	f	1.55	60	25.0	NO	NO	130/76	191	222	6 months	tinea pedis	t2	o
14	35	f	1.6	84	32.8	NO	NO	150/80	119	185	6 months	ulcer foot	t2	o
15	56	f	1.55	65	27.1	NO	NO	130/70	99	198	12	Scleredema	t2	o+i
16	60	f	1.55	60	25.0	NO	NO	120/70	180	360	3	cellulitis,pruritus	t2	o
17	70	f	1.6	50	19.5	NO	NO	120/70	126	226	newly detected	furuncle,pruritus	t2	o
18	65	f	1.6	56	21.9	NO	NO	140/90	198	302	3	tinea corporis	t2	o
19	46	f	1.58	60	24.0	NO	NO	130/80	218	302	2 months	Abscess	t2	o
20	42	f	1.4	45	23.0	NO	NO	150/90	250	398	10	vaginal candidiasis,pruritus	t2	O+I
21	54	f	1.5	60	26.7	NO	NO	120/70	250	350	6	onychomycosis	t2	o
22	55	f	1.6	70	27.3	NO	NO	130/80	240	340	10	Scleredema,pruritus	t2	o
23	36	f	1.6	70	27.3	NO	NO	150/70	265	365	2	skin tag, herpes zoster	t2	o
24	58	f	1.55	60	25.0	NO	NO	130/60	211	312	30	Intertrigo	t2	O+I
25	68	f	1.6	60	23.4	NO	NO	140/90	413	450	1	tinea corporis,pruritus	t2	o
26	64	f	1.55	55	22.9	NO	NO	130/80	218	312	2 months	Eczema	t2	o
27	74	f	1.6	60	23.4	NO	NO	120/70	82	98	10	tinea pedis	t2	o
28	52	f	1.55	50	20.8	NO	NO	140/80	168	268	5	vaginal candidiasis	t2	o
29	55	f	1.55	50	20.8	NO	NO	120/70	236	326	7	intertrigo, paronychia,pruritus	t2	o
30	70	f	1.6	65	25.4	NO	NO	130/80	90	105	5	neuropathic ulcer	t2	o
31	70	f	1.5	50	22.2	NO	NO	120/70	90	140	15	ulcer foot,gangrene	t2	o+i
32	40	F	1.45	60	28.6	NO	NO	130/70	98	156	newly detected	Abscess	t2	o
33	65	F	1.5	50	22.2	NO	NO	130/80	126	246	5	vaginal candidiasis,pruritus	t2	o
34	40	F	1.6	80	31.2	NO	NO	110/70	85	160	1.5	tinea corporis	t2	o
35	45	F	1.52	69	29.9	NO	NO	140/90	136	245	newly detected	tinea corporis	t2	o
36	63	F	1.57	55	22.3	NO	NO	150/90	250	326	15	ulcer foot	t2	o+i
37	32	F	1.6	58	22.7	NO	NO	120/80	116	240	6	vaginal candidiasis	t2	o
38	42	F	1.63	68	25.6	NO	NO	120/80	126	220	5	tinea corporis,paronychia	t2	o
39	46	F	1.54	56	23.6	NO	NO	130/90	110	198	3	Dermopathy	t2	o
40	54	F	1.62	61	23.3	NO	NO	140/90	106	176	11	neuropathic ulcer	t2	o+i
41	35	F	1.7	70	24.2	NO	NO	120/80	138	240	newly detected	Furuncle,skin tags	t2	o
42	45	F	1.54	51	21.5	NO	NO	120/80	135	234	10	ulcer foot,gangrene	t2	o

43	61	F	1.65	75	27.6	NO	NO	140/80	140	280	15	neuropathic ulcer	t2	o+i
44	42	F	1.7	66	22.8	NO	NO	150/90	230	380	5	Eczema	t2	o
45	35	F	1.68	58	20.6	NO	NO	120/80	132	240	newly detected	Acanthosis	t2	o
46	46	F	1.55	52	21.7	NO	NO	140/80	125	210	7	skin tags/acanthosis	t2	o
47	45	F	1.58	59	23.6	NO	NO	160/100	140	260	8	intertrigo	t2	o
48	36	F	1.6	57	22.3	NO	NO	140/80	130	280	2	icthyosis	t2	o
49	38	F	1.51	59	25.9	NO	NO	130/90	112	190	8	icthyosis	t2	o
50	63	F	1.52	55	23.8	NO	NO	130/80	156	302	15	ulcer foot, gangrene	t2	o+i
51	62	F	1.59	58	23.0	NO	NO	130/80	115	202	19	neuropathic ulcer	t2	o+i
52	53	F	1.59	53	21.0	NO	NO	120/84	130	260	8	Scleredema	t2	o
53	36	F	1.52	50	21.7	NO	NO	120/80	145	260	4	vaginal candidiasis	t2	o
54	47	F	1.42	49	24.3	NO	NO	140/90	230	390	7	eczema,pruritus	t2	o
55	45	F	1.6	59	23.1	NO	NO	130/80	150	236	newly detected	acanthosis	t2	o
56	55	F	1.57	53	21.5	NO	NO	120/80	190	420	12	ulcer foot	t2	o
57	42	F	1.61	60	23.2	NO	NO	140/84	140	260	2	tinea pedis	t2	o
58	57	F	1.54	48	20.2	NO	NO	120/80	140	280	8	neuropathic ulcer	t2	o
59	61	F	1.53	55	23.5	NO	NO	140/80	130	270	17	intertrigo, paronychia	t2	o+i
60	60	F	1.52	66	28.6	NO	NO	140/80	190	320	10	ulcer foot	t2	o
61	40	F	1.51	65	28.5	NO	NO	120/88	156	302	newly detected	vaginal candidiasis	t2	o
62	37	F	1.57	70	28.4	NO	NO	120/80	140	280	8	tinea corporis,paronychia	t2	o
63	59	F	1.56	69	28.4	NO	NO	150/90	113	186	9	Scleredema	t2	o
64	58	F	1.6	77	30.1	NO	NO	160/100	155	285	16	ulcer foot,gangrene	t2	o
65	36	F	1.62	65	24.8	NO	NO	120/80	113	202	3	dermopathy	t2	o
66	52	F	1.55	58	24.2	NO	NO	150/70	160	280	9	vaginal candidiasis	t2	o
67	55	F	1.62	75	28.6	NO	NO	140/80	140	270	16	neuropathic ulcer	t2	o
68	66	F	1.57	62	25.2	NO	NO	140/90	128	302	18	ulcer foot	t2	O+I
69	52	F	1.51	64	28.1	NO	NO	120/84	160	237	14	ulcer foot,gangrene	t2	o
70	51	F	1.58	68	27.2	NO	NO	140/80	139	218	9	skin tags/acanthosis	t2	o
71	37	F	1.51	70	30.7	NO	NO	120/84	147	232	newly detected	skin tags	t2	o
72	48	F	1.62	62	23.6	NO	NO	120/80	132	260	7	onychomycosis	t2	o
73	38	F	1.5	69	30.7	NO	NO	120/86	240	420	5	eczema,pruritus	t2	o
74	58	F	1.59	72	28.5	NO	NO	140/80	189	270	16	ulcer foot	t2	o
75	61	F	1.53	68	29.1	NO	NO	140/80	200	290	17	abscess, skin tags	t2	o
76	48	F	1.57	62	25.2	NO	NO	120/86	178	299	2	dermopathy	t2	o
77	37	F	1.5	58	25.8	NO	NO	120/80	168	257	newly detected	vaginal candidiasis,pruritus	t2	o
78	38	F	1.58	51	20.4	NO	NO	140/88	145	257	6	tinea pedis	t2	o
79	47	F	1.59	60	23.7	NO	NO	120/80	170	287	3	tinea corporis	t2	o
80	51	F	1.6	56	21.9	NO	NO	160/100	211	290	10	neuropathic ulcer	t2	o
81	55	F	1.57	72	29.2	NO	NO	120/80	132	230	14	onychomycosis	t2	o+i
82	59	F	1.6	59	23.1	NO	NO	130/90	200	410	13	ulcer foot,paronychia	t2	o
83	39	F	1.55	60	25.0	NO	NO	120/80	189	270	5	tinea pedis	t2	o
84	31	F	1.59	58	23.0	NO	NO	120/80	200	290	newly detected	skin tags, acanthosis	t2	o
85	41	F	1.59	55	21.8	NO	NO	120/88	178	280	4	skin tags, acanthosis	t2	o
86	50	F	1.51	68	29.8	NO	NO	140/90	145	240	10	tinea pedis	t2	o
87	40	F	1.52	74	32.0	NO	NO	120/80	179	256	5	acanthosis	t2	o
88	60	F	1.6	76	29.7	NO	NO	150/90	150	268	17	neuropathic ulcer	t2	o+i
89	38	F	1.53	65	27.8	NO	NO	120/80	156	228	newly detected	cellulitis	t2	o
90	49	F	1.52	70	30.3	NO	NO	140/80	146	260	8	intertrigo, paronychia	t2	o
91	31	F	1.68	75	26.6	NO	NO	130/90	156	279	newly detected	tinea corporis	t2	o
92	61	F	1.63	65	24.5	NO	NO	120/80	187	266	13	vaginal candidiasis	t2	o+i
93	39	F	1.5	60	26.7	NO	NO	120/80	178	269	5	eczema	t2	o

94	48	F	1.52	57	24.7	NO	NO	120/80	189	292	4	intertrigo, paronychia	t2	o
95	60	F	1.62	78	29.7	NO	NO	140/86	179	299	19	neuropathic ulcer	t2	o
96	39	F	1.54	69	29.1	NO	NO	130/88	168	294	6	icthyosis	t2	o
97	61	F	1.52	54	23.4	NO	NO	140/90	156	289	17	skin tags, acanthosis	t2	o+i
98	41	F	1.61	69	26.6	NO	NO	120/80	193	276	2	vaginal candidiasis	t2	o
99	38	F	1.52	57	24.7	NO	NO	120/84	147	269	4	onychomycosis	t2	o
100	47	F	1.51	70	30.7	NO	NO	120/80	189	299	2	eczema	t2	o
101	61	F	1.61	57	22.0	NO	NO	140/84	129	279	18	intertrigo, paronychia	t2	o+i
102	33	F	1.55	60	25.0	NO	NO	120/86	138	280	2	cellulitis	t2	o
103	55	F	1.59	58	23.0	NO	NO	140/90	169	289	9	dermopathy	t2	o
104	37	F	1.66	68	24.7	NO	NO	120/80	178	289	3	abscess	t2	o
105	61	F	1.6	57	22.3	NO	NO	150/100	189	290	11	ulcer foot	t2	o
106	39	F	1.62	72	27.4	NO	NO	120/86	167	277	newly detected	vaginal candidiasis	t2	o
107	42	F	1.55	65	27.1	NO	NO	120/80	180	298	newly detected	abscess	t2	o
108	38	F	1.53	67	28.6	NO	NO	120/84	170	289	newly detected	abscess	t2	o
109	64	F	1.6	52	20.3	NO	NO	120/80	189	268	10	neuropathic ulcer	t2	o
110	59	F	1.5	69	30.7	NO	NO	140/86	178	270	18	ulcer foot	t2	O+I
111	49	F	1.54	55	23.2	NO	NO	140/80	156	287	5	skin tags	t2	o
112	57	F	1.6	75	29.3	NO	NO	160/100	157	298	14	acanthosis	t2	O+I
113	39	F	1.58	64	25.6	NO	NO	120/80	145	270	3	Scleredema	t2	o
114	55	F	1.59	77	30.5	NO	NO	140/80	167	299	10	Furuncle	t2	o
115	49	F	1.58	79	31.7	NO	NO	120/84	189	301	5	Cellulitis	t2	o
116	60	F	1.61	79	30.5	NO	NO	140/88	167	289	17	ulcer foot	t2	O+I
117	47	F	1.51	58	25.4	NO	NO	120/80	150	268	8	skin tags	t2	o
118	38	F	1.6	76	29.7	NO	NO	120/80	156	228	4	dermopathy	t2	o
119	65	F	1.52	79	34.2	NO	NO	160/100	160	260	14	vaginal candidiasis	t2	o+i
120	49	F	1.55	78	32.5	NO	NO	140/80	149	279		cellulitis	t2	o
121	41	F	1.51	75	32.9	NO	NO	120/84	187	280	newly detected	Furuncle	t2	o
122	62	F	1.63	88	33.1	NO	NO	130/90	178	269	18	neuropathic ulcer	t2	o
123	53	F	1.59	77	30.5	NO	NO	120/80	189	292	13	Furuncle	t2	o
124	49	F	1.6	68	26.6	NO	NO	130/84	179	299	10	Cellulitis	t2	o
125	40	F	1.57	53	21.5	NO	NO	120/80	168	300	9	vaginal candidiasis	t2	o
126	42	m	1.68	70	24.8	YES	YES	130/70	140	230	10	Eczema	t2	o
127	56	m	1.72	75	25.4	YES	YES	110/70	156	220	12	ulcer foot	t2	o
128	43	m	1.66	77	28.0	NO	YES	130/80	136	245	15	neuropathic ulcer	t2	o
129	44	m	1.69	70	24.5	NO	NO	124/60	250	326	10 months	balanoposthitis	t2	o
130	58	m	1.63	65	24.5	NO	NO	150/100	150	275	7	Furuncle	t2	o
131	52	m	1.75	80	26.1	YES	NO	140/90	126	226	8	tinea pedis	t2	o
132	51	m	1.55	75	31.2	NO	NO	160/100	142	282	5	Eczema	t2	o
133	50	m	1.77	68	21.7	YES	YES	130/80	232	322	4	ulcer foot	t2	o
134	48	m	1.68	76	26.9	NO	NO	120/70	155	260	6	Abscess	t2	o
135	42	m	1.65	75	27.6	NO	YES	120/70	145	280	3	onychomycosis	t2	o
136	53	m	1.7	79	27.3	NO	NO	130/90	190	270	newly detected	balanoposthitis	t2	o
137	40	m	1.64	70	26.0	YES	YES	140/70	186	276	10	ulcer foot	t2	o
138	55	m	1.59	72	28.5	NO	YES	150/90	143	283	6	neuropathic ulcer	t2	o
139	44	m	1.63	65	24.5	YES	YES	140/90	202	402	4	Icthyosis	t2	o
140	59	m	1.75	70	22.9	YES	YES	130/90	178	302	12	balanoposthitis	t2	o
141	60	m	1.7	65	22.5	YES	YES	120/90	162	280	7 months	neuropathic ulcer	t2	o
142	43	m	1.65	66	24.3	NO	NO	110/80	156	276	5	Scleredema	t2	o
143	49	m	1.57	68	27.6	NO	NO	170/100	180	302	8	Abscess	t2	o
144	54	m	1.67	70	25.1	NO	NO	150/90	136	246	6	balanoposthitis	t2	o
145	50	m	1.74	74	24.5	NO	YES	140/90	128	220	10	neuropathic ulcer	t2	o

146	51	m	1.64	78	29.0	NO	YES	130/80	126	280	5 months	ulcer foot	t2	o
147	44	m	1.68	76	26.9	NO	NO	130/80	146	300	9	Furuncle	t2	o
148	56	m	1.7	80	27.7	NO	NO	120/84	136	276	10	Abscess	t2	o
149	55	m	1.75	78	25.5	NO	NO	140/90	140	270	7 months	balanoposthitis	t2	o
150	41	m	1.72	68	23.0	NO	YES	140/80	190	320	11	neuropathic ulcer	t2	o
151	59	m	1.7	65	22.5	NO	NO	130/80	163	297	newly detected	intertrigo, paronychia	t2	o
152	52	m	1.65	66	24.3	NO	YES	120/70	165	276	8	ulcer foot	t2	o
153	51	m	1.6	63	24.6	YES	YES	120/80	160	296	5	balanoposthitis	t2	o
154	43	m	1.72	62	21.0	NO	NO	130/90	140	280	8	Abscess	t2	o
155	50	m	1.68	76	26.9	NO	YES	130/80	126	254	8	neuropathic ulcer	t2	o
156	59	m	1.6	64	25.0	NO	YES	120/90	158	278	4	intertrigo, paronychia	t2	o
157	43	m	1.74	70	23.1	NO	NO	120/80	152	290	8	Cellulitis	t2	o
158	48	m	1.7	73	25.3	NO	NO	150/100	138	240	6	Eczema	t2	o
159	56	m	1.6	65	25.4	NO	YES	160/90	129	260	5	ulcer foot	t2	o
160	51	m	1.65	75	27.6	YES	YES	130/80	126	320	4	skin tag	t2	o
161	45	m	1.68	69	24.5	YES	YES	160/70	180	310	10	Cellulitis	t2	o
162	60	m	1.63	80	30.1	NO	NO	160/80	184	340	12	ulcer foot	t2	o
163	55	m	1.73	76	25.4	YES	YES	130/90	188	360	13	onychomycosis,pruritus	t2	o
164	58	m	1.62	78	29.7	YES	YES	120/80	143	283	6	neuropathic ulcer	t2	o
165	56	m	1.64	74	27.5	NO	NO	120/80	146	260	2	xanthoma	t2	o
166	52	m	1.65	75	27.6	NO	NO	130/80	125	226	1	balanoposthitis	t2	o
167	48	m	1.63	73	27.5	NO	NO	140/90	170	290	5	Eczema	t2	o
168	50	m	1.6	72	28.1	NO	NO	150/90	180	300	4	abscess,pruritus	t2	o
169	52	m	1.58	71	28.5	NO	NO	150/70	129	290	6	balanoposthitis	t2	o
170	62	m	1.78	70	22.1	NO	NO	126/80	149	289	7	Cellulitis	t2	o
171	65	m	1.75	68	22.2	NO	NO	110/80	202	315	15	Abscess	t2	o
172	65	m	1.74	80	26.4	YES	YES	120/70	190	340	12	neuropathic ulcer	t2	o
173	60	m	1.72	75	25.4	YES	YES	130/90	140	260	13	ulcer foot	t2	o
174	62	m	1.68	75	26.6	YES	YES	140/90	180	270	10	neuropathic ulcer	t2	o
175	58	m	1.6	73	28.5	YES	YES	150/100	180	260	8 months	ulcer foot	t2	o
176	45	m	1.61	65	25.1	NO	NO	120/80	130	280	newly detected	Abscess	t2	o
177	43	m	1.68	67	23.7	NO	YES	130/70	140	270	10	neuropathic ulcer	t2	o
178	56	m	1.7	79	27.3	YES	YES	120/90	150	259	12	ulcer foot	t2	o
179	67	m	1.69	75	26.3	NO	NO	140/80	142	302	9	abscess, skin tags	t2	o
180	64	m	1.64	68	25.3	NO	NO	136/78	162	320	6	Cellulitis	t2	o
181	54	m	1.72	79	26.7	NO	NO	110/70	136	245	3	tinea corporis	t2	o
182	45	m	1.76	80	25.8	YES	YES	136/80	156	298	12	neuropathic ulcer	t2	o
183	58	m	1.67	70	25.1	NO	NO	120/80	126	238	2	balanoposthitis	t2	o
184	46	m	1.75	77	25.2	NO	NO	150/100	236	286	7	Furuncle	t2	o
185	48	m	1.63	72	27.1	NO	NO	140/90	280	340	6	Cellulitis	t2	o
186	49	m	1.72	79	26.7	NO	NO	160/90	124	224	8	Icthyosis	t2	o
187	60	m	1.65	69	25.4	YES	YES	146/78	234	320	10	ulcer foot	t2	o
188	58	m	1.72	74	25.0	NO	NO	110/80	132	230	9	balanoposthitis	t2	o
189	57	m	1.74	76	25.1	NO	NO	120/90	142	265	5	Eczema	t2	o
190	45	m	1.78	77	24.3	NO	NO	130/90	213	289	4	carbuncle	t2	o
191	42	m	1.8	80	24.7	NO	NO	120/80	215	321	6	Intertrigo	t2	o
192	47	m	1.69	69	24.2	NO	YES	140/80	221	312	10	neuropathic ulcer	t2	o+i
193	59	m	1.63	68	25.6	YES	NO	150/90	245	333	13	cellulitis,pruritus	t2	o+i
194	56	m	1.71	70	23.9	NO	NO	160/90	276	343	5	intertrigo,pruritus	t2	o
195	48	m	1.66	76	27.6	NO	YES	150/80	265	354	11	neuropathic ulcer,pruritus	t2	O+I
196	47	m	1.75	83	27.1	NO	NO	126/90	143	232	1	carbuncle	t2	o
197	42	m	1.62	72	27.4	NO	YES	130/70	165	254	15	ulcer foot	t2	o+i
198	60	m	1.78	78	24.6	YES	YES	150/90	176	264	13	neuropathic ulcer	t2	o+i
199	61	m	1.72	76	25.7	NO	NO	140/80	189	276	5	Eczema	t2	o

200	49	m	1.69	80	28.0	YES	YES	130/70	187	278	11	neuropathic ulcer	t2	o+i
201	65	m	1.78	82	25.9	YES	YES	140/90	165	276	5	Scleredema	t2	o
202	53	m	1.74	79	26.1	NO	NO	160/90	132	245	4	skin tags	t2	o
203	44	m	1.68	78	27.6	NO	NO	140/78	143	265	3	Intertrigo	t2	o
204	47	m	1.65	76	27.9	NO	NO	110/80	176	256	8	onychomycosis	t2	o
205	56	m	1.76	75	24.2	NO	NO	120/70	134	254	8	Vitiligo	t2	o
206	48	m	1.75	80	26.1	NO	NO	130/70	123	224	7	Intertrigo	t2	o
207	59	m	1.78	76	24.0	NO	YES	140/90	156	246	10	ulcer foot	t2	o+i
208	60	m	1.68	77	27.3	NO	YES	150/80	176	254	10	neuropathic ulcer	t2	o+i
209	57	m	1.62	70	26.7	NO	NO	112/70	145	265	8	Icthyosis	t2	o
210	58	m	1.73	76	25.4	NO	NO	114/60	232	280	2	carbuncle	t2	o
211	44	m	1.69	80	28.0	NO	NO	124/80	200	280	1	abscess, skin tags	t2	o
212	48	m	1.78	80	25.3	NO	NO	148/80	198	270	5	tinea pedis	t2	o
213	59	m	1.72	70	23.7	NO	NO	150/80	178	260	6	onychomycosis	t2	o
214	61	m	1.66	80	29.0	NO	NO	146/90	167	270	6	Abscess	t2	o
215	64	m	1.64	82	30.5	NO	NO	150/80	154	232	8	Cellulitis	t2	o
216	56	m	1.62	83	31.6	NO	NO	134/80	132	198	2	Cellulitis	t2	o
217	41	m	1.65	70	25.7	NO	NO	130/70	155	262	10	Eczema	t2	o
218	61	m	1.68	75	26.6	NO	NO	120/80	143	263	5	Icthyosis	t2	O
219	44	m	1.72	78	26.4	NO	NO	140/80	148	196	4	xanthoma	t2	O
220	48	m	1.68	73	25.9	YES	YES	150/90	134	200	5	tinea corporis	t2	O
221	55	m	1.72	76	25.7	NO	NO	120/80	150	250	2	Cellulitis	t2	O
222	43	m	1.8	80	24.7	NO	NO	150/90	132	342	6	Abscess	t2	O
223	56	m	1.76	67	21.6	NO	NO	140/70	176	266	7	Eczema	t2	O
224	57	m	1.76	76	24.5	NO	NO	150/80	143	270	6	Abscess	t2	O
225	48	m	1.64	79	29.4	NO	YES	116/76	123	260	4	Cellulitis	t2	O
226	19	f	1.6	81	19.5	NO	NO	110/70	167	320	3	tinea pedis	t1	I
227	20	f	1.58	54	21.6	NO	NO	112/80	155	240	7	dry scaly palms	t1	I
228	30	f	1.48	50	22.8	NO	NO	110/60	177	160	10	Icthyosis	t1	i
229	29	f	1.52	51	22.1	NO	NO	120/70	166	268	9	keratosis pilaris	t1	i
230	18	f	1.62	55	21.0	NO	NO	110/80	161	251	7	Icthyosis	t1	i
231	21	f	1.58	52	20.8	NO	NO	120/70	132	251	4	Vitiligo	t1	i
232	20	f	1.6	50	19.5	NO	NO	130/70	101	196	5	Scleroderma like changes	t1	i
233	20	f	1.65	50	18.4	NO	NO	132/60	92	176	6	icthyosis	t1	i
234	28	f	1.54	50	21.1	NO	NO	130/70	96	185	6	tinea pedis	t1	i
235	25	f	1.62	58	22.1	NO	NO	120/74	152	250	5	dry scaly palms	t1	i
236	30	f	1.65	55	20.2	NO	NO	120/70	104	184	8	scleroderma like changes	t1	i
237	22	f	1.5	46	20.4	NO	NO	120/80	110	198	4	keratosis pilaris	t1	i
238	19	f	1.55	52	21.6	NO	NO	130/90	132	252	9	scleroderma like changes	t1	i
239	25	f	1.6	48	18.7	NO	NO	120/70	106	186	7	icthyosis	t1	i
240	16	f	1.55	48	20.0	NO	NO	120/80	300	420	8	scleroderma like changes	t1	i
241	26	m	1.7	65	22.5	NO	NO	100/60	140	240	8	icthyosis	t1	i
242	25	m	1.65	70	25.7	NO	NO	120/70	120	190	7	keratosis pilaris	t1	i
243	24	m	1.7	68	23.5	NO	NO	130/70	160	270	10	icthyosis	t1	i
244	18	m	1.74	68	22.5	NO	NO	180/50	132	232	3	scleroderma like changes	t1	i
245	26	m	1.75	65	21.2	NO	NO	110/70	118	198	6	dry scaly palms	t1	i
246	30	m	1.68	65	23.0	YES	NO	130/80	152	262	9	icthyosis	t1	i
247	29	m	1.6	60	23.4	NO	NO	130/70	143	263	9	scleroderma like changes	t1	i
248	20	m	1.66	62	22.5	NO	NO	126/70	112	196	7	icthyosis	t1	i
249	20	m	1.7	60	20.8	NO	NO	128/70	108	158	8	icthyosis	t1	i
250	32	m	1.68	60	21.3	YES	NO	110/70	150	250	12	scleroderma like changes	t1	i

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : An Observational study on Mucocutaneous manifestations of type 1 and type 2 diabetes mellitus..

Principal Investigator : Dr. T Jaya Packiam

Designation : PG in MD (General Medicine)

Department : Department of (General Medicine) Government Stanley Medical College, Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 02.07.2014 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

T_1 & T_2 சர்க்கரை வியாதியினால் தோலில் ஏற்படும் மாற்றங்கள் & நோய்கள் குறித்த ஆய்வு

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை
சென்னை - 600 001.

பங்கு பெறும் நோயாளியின் பெயர் : வயது

நோயாளியின் விலாசம் : பாலினம் : ஆண் ☐ பெண் ☐

நோயாளி இதனை ( ) குறிக்கவும் :

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் என்னை இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்க அனுமதிக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் என்னை இவ்வாய்வில் இருந்து விலக்கிக் கொள்ளலாம் என்று அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவும் மேலும் இதை சார்ந்த ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கையை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன் என்னை ஆய்வில் விலக்கிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடத்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதி அளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய் குறி தென்பட்டாலோ உடனே அதனை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன்

நோயாளியின் கையொப்பம் ..... இடம் ..... தேதி .....

கட்டை விரல் (இந்த படிவம் படித்து காட்டப்பட்டு புரிந்து கைரேகை அளிக்கிறேன்)

பங்கு பெறுபவர்களின் பெயர் மற்றும் விலாசம் .....

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ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

..... ஆய்வாளரின் பெயர் .....

தகவல் படிவம்

மதிப்பிற்குரிய அய்யா / அம்மையீர்

T_1 & T_2 சர்க்கரை வியாதியினால் தோலில் பல மாற்றங்கள் & நோய்கள் ஏற்படுகின்றன. இந்த மாற்றங்கள் இரத்த சர்க்கரையின் அளவு மற்றும் காலத்தைப் பொறுத்து மாறுபடுகிறது. அதனை கண்டறியதற்காக இந்த ஆய்வு மேற்கொள்ளப்படுகிறது.

அதற்கு உங்கள் முழு சம்மதம் வேண்டியே இந்த படிவம் வழங்கப்படுகிறது. தாங்கள் இந்த ஆய்வில் பங்கேற்க சம்மதிக்கும் பட்சத்தில் இந்த படிவத்தை முழுவதும் படித்துப்பார்த்து முழு மனதுடன் ஒப்புதல் அளிக்க கையொப்பமிடுமாறு கேட்டுக் கொள்கிறேன்.

நோயாளி/உறவினரின் கையொப்பம்
இடது பெருவிரல் ரேகை
(மருத்துவரால் படித்துக்காட்டப்பட்டது)